"The prognostic value of T peak – T end interval on the surface Electrocardiogram in patients undergoing reperfusion therapy for ST-segment Elevation Myocardial Infarction"

A DISSERTATION SUBMITTED TO THE

DR. MGR MEDICAL UNIVERSITY, CHENNAI, TAMIL NADU

IN PARTIAL FULFILLMENT OF DM BRANCH II-CARDIOLOGY

EXAMINATION TO BE HELD IN AUGUST 2014

Certificate

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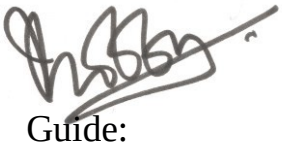

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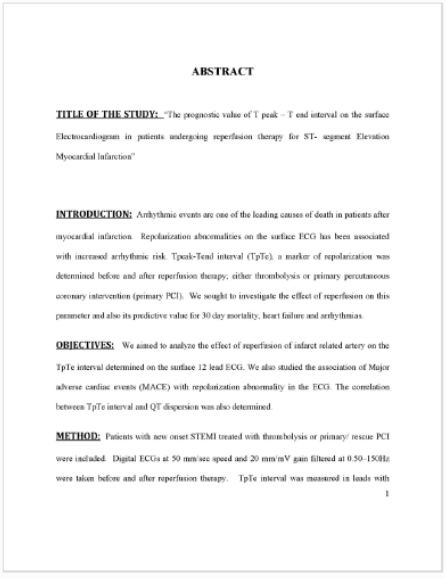

ABSTRACT

TITLE OF THE STUDY: "The prognostic value of T peak – T end interval on the surface Electrocardiogram in patients undergoing reperfusion therapy for ST- segment Elevation Myocardial Infarction"

INTRODUCTION: Arrhythmic events are one of the leading causes of death in patients after myocardial infarction. Repolarization abnormalities on the surface ECG has been associated with increased arrhythmic risk. Tpeak-Tend interval (TpTe), a marker of repolarization was determined before and after reperfusion therapy; either thrombolysis or primary percutaneous coronary intervention (primary PCI). We sought to investigate the effect of reperfusion on this parameter and also its predictive value for 30 day mortality, heart failure and arrhythmias.

OBJECTIVES: We aimed to analyze the effect of reperfusion of infarct related artery on the TpTe interval determined on the surface 12 lead ECG. We also studied the association of Major adverse cardiac events (MACE) with repolarization abnormality in the ECG. The correlation between TpTe interval and QT dispersion was also determined.

METHOD: Patients with new onset STEMI treated with thrombolysis or primary/ rescue PCI were included. Digital ECGs at 50 mm/sec speed and 20 mm/mV gain filtered at 0.50-150Hz were taken before and after reperfusion therapy. TpTe interval was measured in leads with

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September 30, 2013

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Sub: **FLUID Research grant project:**
The prognostic value of Tpeak-Tend interval on the surface Electrocardiogram in patients undergoing reperfusion therapy for ST-segment elevation Myocardial Infarction.
Dr. Subhrangshu Dey, Senior Registrar, Dept of Cardiology, Dr Bobby John, Cardiology.

Ref: IRB Min. No. 8291 [OBSERVE] dated 16.04.2013

Dear Dr. Subhrangshu Dey,

The Institutional Review Board (Blue, Research and Ethics Committee) of the Christian Medical College, Vellore, reviewed and discussed your project entitled "The prognostic value of Tpeak-Tend interval on the surface Electrocardiogram in patients undergoing reperfusion therapy for ST-segment elevation Myocardial Infarction." on April 16, 2013.

The Committees reviewed the following documents:

1. Format for application to IRB submission
2. Patient Information Sheet and Informed Consent Form (English, Tamil and Telugu)
3. Proforma
4. Cvs of Drs. Subhrangshu Dey, Bobby John.
5. A CD containing documents 1 – 4

The following Institutional Review Board (Research & Ethics Committee) members were present at the meeting held on April 16, 2013 in the CREST/SACN Conference Room, Christian Medical College, Bagayam, Vellore 632002.

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We approve the project tbe conducted as presented.

The Institutional EthifS Committee expects to be informed about the progress of the project, any adverse events occurring in the course of the project, any amendments in the protocol and thpat,ent information f inf rmed consent. On completion of the study you are expe,cted to submit a copy of the final re ort. Respective forms can be downloa ed from the following link: http:/1172.16.11.136/Research/JRB Polices.btml in the GMC Intranet and in the CMC website link address: http: NW\yw.cmch"vellore.edu /statje/researcb/Jndex.btml.

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A sum of 15,650/- INR (Rupees Fifteen Thousand Six Hundred and Fifty only) will be granted for 12 months.

Yours sincerely

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Institutional Review Board

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I express my sincere thanks to all the patients who participated in this study without whom this research would not have been possible.

And above all, I thank The Almighty for everything.

Contents

ABSTRACT

TITLE OF THE STUDY: "The prognostic value of T peak – T end interval on the surface Electrocardiogram in patients undergoing reperfusion therapy for ST- segment Elevation Myocardial Infarction"

INTRODUCTION: Arrhythmic events are one of the leading causes of death in patients after myocardial infarction. Repolarization abnormalities on the surface ECG has been associated with increased arrhythmic risk. We sought to investigate the effect of reperfusion, on Tpeak-Tend interval (TpTe), a marker of repolarization and also its predictive value for 30 day mortality, heart failure and arrhythmias.

OBJECTIVES: We aimed to analyze the effect of reperfusion of infarct related artery on the TpTe interval determined on the surface 12 lead ECG. We also studied the association of Major adverse cardiac events (MACE) with repolarization abnormality in the ECG. The correlation between TpTe interval and QT dispersion was also determined.

METHODS: Patients with new onset STEMI treated with thrombolysis or primary/ rescue PCI were included. Digital ECGs at 50 mm/sec speed and 20 mm/mV gain filtered at 0.50–150Hz were taken before and after reperfusion therapy. TpTe interval was measured in leads with limited ST-segment deviation and so also the QTc. Echocardiography was done before hospital

discharge for all patients. Angiographic parameters of patients undergoing primary or rescue PCI were recorded. All patients were followed up at 30 days.

RESULTS: From June 2013 to December 2013, total of 216 patients were included of which 183 were males (85.1%). The mean age was 54.86 years (range 24-80 years). One hundred and thirteen patients underwent primary PCI (52.3%), 57 underwent lysis (26.4%) and remaining 46(21.3%) had rescue PCI. Thirty day Mortality was 5.1 % (11 patients).

The median pre TpTe interval was 84.5ms and the 25^{th}, 50^{th} and 75^{th} percentiles were 80, 84 and 100 ms respectively. The median post TpTe intervals were 76.7ms (64, 76.7 and 80ms), 75ms (60, 75 and 80ms) and 73.3ms (66.7, 73.3 and 80ms) respectively in the primary PCI, thrombolysis and rescue PCI groups. There was statistically significant reduction in TpTe interval at 90 minutes following reperfusion (p values of 0.0001, 0.0001 and 0.004 respectively). This reduction was uniformly seen in all the treatment arms.

Of the 216 patients, 210 were followed up at 30 days. Six patients were lost to follow up. Eleven patients died 11(5.1%) patients had died. The pre TpTe interval of more than 100 ms was associated with increased risk of ventricular arrhythmias (OR 13.21, 95% CI 1.16 – 150.57). However, it did not predict mortality at 30 days (OR – 1.405, 95% CI – 0.288 – 6.842) or heart failure in the 8 patients at follow up. (OR 2.14, 95% CI 0.412-11.148). There was no correlation between the TpTe interval and QTc dispersion. After adjusting for established risk factors, TpTe interval difference (pre – post) was found to be significantly associated with duration of chest pain and Killip class.

CONCLUSION: In patients with STEMI undergoing reperfusion, the TpTe interval was significantly reduced after reperfusion therapy (either primary PCI or thrombolysis). Pre-reperfusion TpTe predicted the risk of arrhythmias at 30 days. However, it did not predict subsequent all cause mortality and heart failure at 30 days. QT dispersion did not correlate with changes in TpTe interval at 90 minutes following reperfusion.

CHAPTER 1

INTRODUCTION

The modern “reperfusion era” of coronary care was introduced by the intra-venous fibrinolysis, and with increased use of aspirin supplemented by development of primary percutaneous coronary intervention, the case fatality rate of STEMI has reduced significantly.(1,2) However, it continues to be a major public health problem in the industrialized world and is slated to rise in the developing countries.(3)

The aim of treatment of STEMI is mechanical reperfusion. In this context, mechanical reperfusion by primary percutaneous coronary intervention (primary PCI) has become the first standard option for the treatment of STEMI, over thrombolysis.(4)

One of the important long term goals of reperfusion strategy is to prevent sudden cardiac death which is most often related to ventricular arrhythmias. Patients with abnormal repolarization have been shown to have increased risk of sudden death.(5) Repolarization of the myocardium is dependent on the perfusion.(6)

Towards this end, electrophysiological characterizations of post-STEMI patients by the indices of repolarization on the surface electrocardiogram (ECG) have shown clinical promise for the prediction of death and malignant arrhythmias. In the standard 12 lead ECG, the interval from the peak (Tp) to the end (Te) of the T wave (TpTe) has been proposed to represent repolarization dispersion in the heart.(7) The corrected QT interval dispersion (CQTD) is another index of

repolarization on the 12 lead ECG.(8) It is the difference between the maximum and the minimum QT duration on the 12 lead surface ECG, corrected to the heart rate.

RELEVANCE OF THE STUDY

There have been very few studies which have looked at TpTe post primary PCI in patients with STEMI and none that have looked at the effect of thrombolysis on TpTe. Previous studies have shown varying results of the mechanical reperfusion of the infarct-related arteries on CQTD. (5, 9)

The purpose of the present study is to analyze prospectively, in patients with STEMI undergoing reperfusion therapy, the acute effects of the reperfusion of the infarct related artery on the TpTe and the CQTd. It will also analyze the effect of reperfusion on this parameter and also its predictive value for 30 day mortality. The study will also compare the results of reperfusion therapy (primary PCI versus thrombolysis) on repolarization indices.

CHAPTER 2

OBJECTIVES

- To analyze prospectively, in patients with STEMI undergoing reperfusion therapy, the effect of reperfusion on the Tpeak-Tend interval on the surface 12 lead Electrocardiogram.
- To study the association of Major Adverse Cardiac with repolarization abnormality in the ECG.
- To study the correlation between TpTe interval and QT dispersion.

CHAPTER 3

REVIEW OF LITERATURE

DEFINITION OF STEMI

The World Health Organization and American Heart Association have given a revised definition for Myocardial Infarction. Either of the following criteria satisfies the diagnosis for acute, evolving or recent STEMI:

1) Typical rise and/or fall of biochemical markers of myocardial necrosis with at least one of the following :
    a. Ischemic symptoms
    b. Development of pathologic Q waves in the ECG
    c. Electrocardiographic changes indicative of ischaemia (ST segment elevation or depression)
    d. Imaging evidence of new loss of myocardium or new regional wall motion abnormality
2) Pathologic findings of an acute Myocardial Infarction

RISK STRATIFICATION AFTER STEMI

Risk stratification after STEMI occurs in several stages – initial presentation, in-hospital course and at the time of hospital discharge (Figure 3.1 and Table 3.1).

Certain demographic and historical factors portends a worse prognosis in patients with STEMI, which includes (5,11–14)

- Female gender
- Age > 65 years
- History of Diabetes Mellitus (>40% increase in adjusted risk of death at 30 days)
- Prior angina pectoris
- Previous MI
- Development of Heart failure after MI entails a higher risk of sudden cardiac death
- Recurrent ischaemia and infarction following STEMI
- Anterior wall (compared to inferior wall)
- RV infarction complicating inferior wall STEMI (compared to inferior wall alone)
- Multiple leads showing ST-segment elevation and a high sum of ST-segment elevation
- Persistent advanced heart block (type II second-degree or third-degree AV block)
- New intraventricular conduction disturbances (bifascicular or trifascicular)
- Persistent horizontal or down sloping ST-segment depression
- Q waves in multiple leads
- ST-segment depressions in anterior leads in patients with inferior MI

- Atrial arrhythmias, especially AF
- Patients with abnormal repolarization

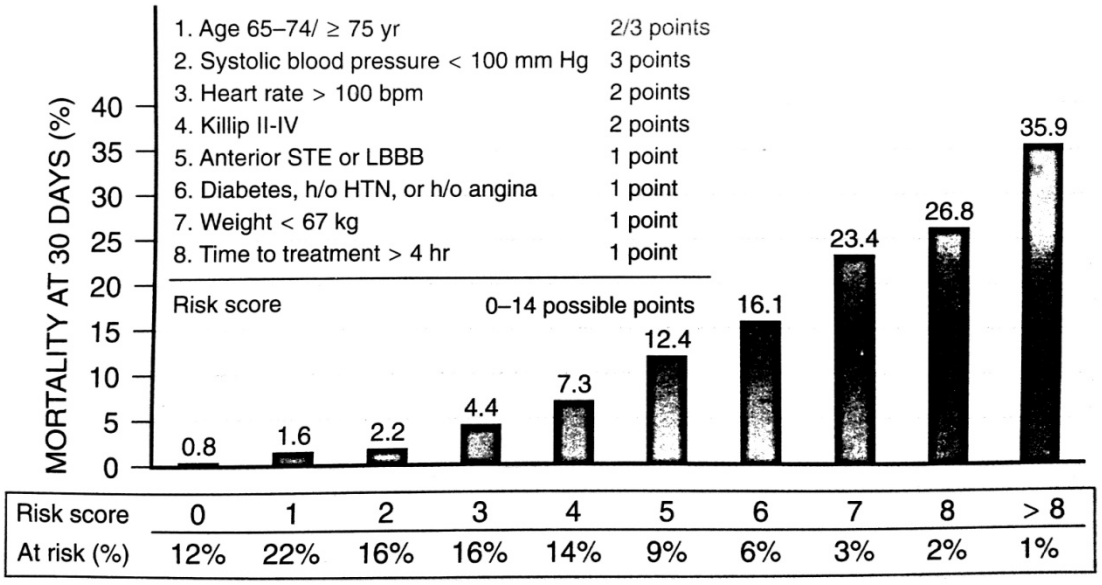
The **TIMI (Thrombolysis for Myocardial Infarction) risk score** for predicting 30-day mortality is as shown in Table 3.1 & Figure 3.1.(15)

TABLE 3. 1- THROMBOLYSIS FOR MYOCARDIAL INFARCTION (TIMI) RISK SCORE

Variable	**Risk Score**
Age 65-74/>75 years	2/3 points
Systolic BP <100 mm Hg	3 points
Heart rate >100 bpm	2 points
Killip class 2-4	2 points
Anterior STEMI or LBBB	1 point
Diabetes, Hypertension, h/o angina	1 point
Weight <67 kg	1 point
Time to treatment>4 hours	1 point

Depending on the risk score, the 30 day Mortality varies.

FIGURE 3. 1-TIMI RISK SCORE DEPICTING 30 DAY MORTALITY FOR ST SEGMENT ELEVATION MYOCARDIAL INFARCTION



Courtesy: Morrow DA, Antman EM, Charlesworth A, Cairns R, Murphy SA, de Lemos JA, et al. TIMI risk score for ST-elevation myocardial infarction: A convenient, bedside, clinical score for risk assessment at presentation: An intravenous nPA for treatment of infarcting myocardium early II trial substudy. Circulation. 2000 Oct 24;102(17):2031–7

Both short- and long-term survival after STEMI depend on three factors(5,16):

1) Resting LV function(most important)
2) Residual potentially ischemic myocardium
3) Susceptibility to serious ventricular arrhythmias

STEMI AND REPERFUSION THERAPY

STEMI is a major cause of mortality and morbidity. In randomized trials, the short-term mortality rate in patients who receive aggressive pharmacologic reperfusion therapy is 6.5-7.5% (17). However, observational data bases suggest that mortality rate of STEMI patients in the community is 15-20%.(18) Considerable variation exists in the management and outcomes of patients with STEMI.(19)

Almost all STEMIs are due to coronary atherosclerosis, usually with superimposed coronary thrombosis. Plaque disruption in the culprit vessel produces complete occlusion of the infarct-related artery. As there is progressive loss of functioning myocytes with persistent occlusion of the infarct-related artery in STEMI, restoration of blood flow to the infarct zone is of paramount importance. Otherwise, left ventricular dilation and ultimate death ensues through a combination of pump failure and electrical instability. Early reperfusion shortens the duration of coronary occlusion, minimizes the degree of ultimate left ventricular dilation and dysfunction and reduces the probability that the STEMI patient will develop pump failure and malignant ventricular tachyarrhythmias.

It is generally accepted that primary PCI is the preferred option for reperfusion, provided it can be delivered in a timely fashion by an experienced operator(>75 PCI procedures/year) and team (at least 200 PCI procedures/year, including at least 36 primary PCI procedures/year).(20) Although late spontaneous reperfusion occurs in some patients, thrombotic occlusion persists in most patients of STEMI.

The goal of treatment of STEMI is myocardial reperfusion.(21) Mechanical reperfusion by primary PCI has become the first choice of treatment. The successful recanalization of the epicardial coronaries are important, but the microvascular flow determines the amount of myocardium salvaged and long term prognosis.

An invasive strategy (primary PCI) is generally preferred if (20) :

1. Skilled PCI laboratory is available with surgical backup
    a. Medical contact-to-balloon or door-to-balloon <90 minutes
    b. (Door-to-balloon) - (Door-to-needle) time <60 minutes
2. High risk from STEMI
    a. Cardiogenic shock
    b. Killip class 3 or 4
3. Contraindications to fibrinolysis, including increased risk of bleeding and ICH
4. Late presentation, i.e., symptom onset > 3 hours ago
5. Diagnosis of STEMI is in doubt

Each 30-minute delay from the symptom onset to PCI increases the relative risk (RR) of 1-year mortality by 8%. (22)

MEASURES OF REPERFUSION

Several techniques can evaluate the adequacy of myocardial perfusion. Electrocardiographic ST-segment resolution (STR) is a strong predictor of outcome in STEMI patients.(16) ST segment resolution reflects flow at the microvascular level and not epicardial coronary flow, and therefore provides better prognostic information than angiogram alone. The absence of early STR after primary PCI identifies patients with higher risk of LV dysfunction and mortality, presumably due to microvascular damage. The 12 lead ECG is a marker of the biologic integrity of myocytes in the infarct zone and can reflect inadequate myocardial perfusion, even in the presence of TIMI grade III flow.

To provide a level of standardization, most investigators describe the flow in the infarct vessel according to Thrombolysis in Myocardial Infarction (TIMI) trial grading system (23):

- Grade 0 - Complete occlusion of the infarct-related artery
- Grade 1 - Some penetration of the contrast material beyond the point of obstruction, but without perfusion of the distal coronary bed
- Grade 2 – Perfusion of the entire infarct vessel into the distal bed but with delayed flow compared with a normal artery
- Grade 3 – Full perfusion of the infarct vessel with normal flow

TIMI grade 3 flows is far superior to grade 2 with regards to (21)

- Infarct size reduction
- Short- and long-term mortality benefit

Therefore, TIMI grade 3 is the goal targeted while opening up an infarct-related artery by primary PCI. (24)

In patients with STEMI, reperfusion therapy aims to improve actual myocardial reperfusion in the infarct zone. Normalization of myocardial perfusion can be impeded by microvascular damage and reperfusion injury.

An angiographic method for assessing myocardial perfusion is the TMP grade (TIMI myocardial perfusion grade developed by Gibson and coworkers). Abnormal TMP grade correlate with mortality risk even in the presence of TIMI 3 flow or a normal TIMI frame count.(25)

- TMP grade 0 – No or minimal blush
- TMP grade 1 – Stain present, blush persists on next injection
- TMP grade 2 – Contrast strongly persistent at the end of washout, gone by next injection
- TMP grade 3 – Normal ground glass appearance of blush, contrast mildly persistent at end of washout

SUDDEN CARDIAC DEATH POST STEMI

After STEMI, patients are at greatest risk of sudden cardiac death caused by malignant ventricular arrhythmias over the first 1-2 years after the index event.(26) Several techniques have been proposed to risk stratify patients, which include the following

- QT dispersion (variability of QT intervals between ECG leads)
- Holter monitoring
- Invasive electrophysiological testing
- Recoding signal-averaged ECG (a measure of delayed, fragmented conduction in the infarct zone)
- Measuring heart rate variability (beat to beat variability in R-R intervals)
- Baroreflex sensitivity (slope of a line relating beat to beat change in sinus rate in response to alteration of blood pressure)
- Increased ventricular ectopic activity

However, these have not proved sufficiently useful in routine clinical practice.(5) The low positive predictive value (<30%) for the noninvasive tests limits their usefulness when viewed in isolation.(27)

EFFECT OF STEMI ON REPOLARIZATION

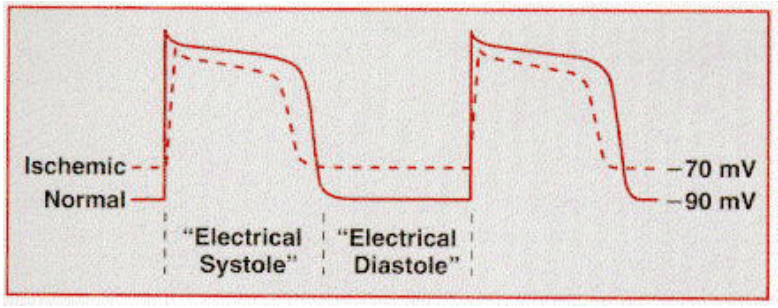
ST-T WAVE ABNORMALITIES

Acute myocardial injury has marked effects on myocardial repolarization. Under normal conditions, the ST segment is nearly isoelectric.(28) This happens as almost all healthy myocardial cells attain approximately the same potential during the initial to middle phases of repolarization, that is, to the plateau phase of the ventricular action potential.

Ischemia has complex time-dependent effects on the electrical properties of myocardial cells.(28) The earliest ECG finding during acute severe ischemia is ST-segment deviation due to a current of injury (both diastolic and systolic). (27,28) Severe acute ischemia reduces the resting membrane potential, abbreviates the duration of action potential in the ischemic area, and decreases the rate of rise and amplitude of phase 0. All these changes cause a voltage gradient between the normal and ischemic zones and leads to current flow between these regions (Figure 3.2). These resulting currents of injury are represented on the surface ECG by ST deviation.(31)

FIGURE 3.2 ELECTRICAL SYSTOLE AND DIASTOLE

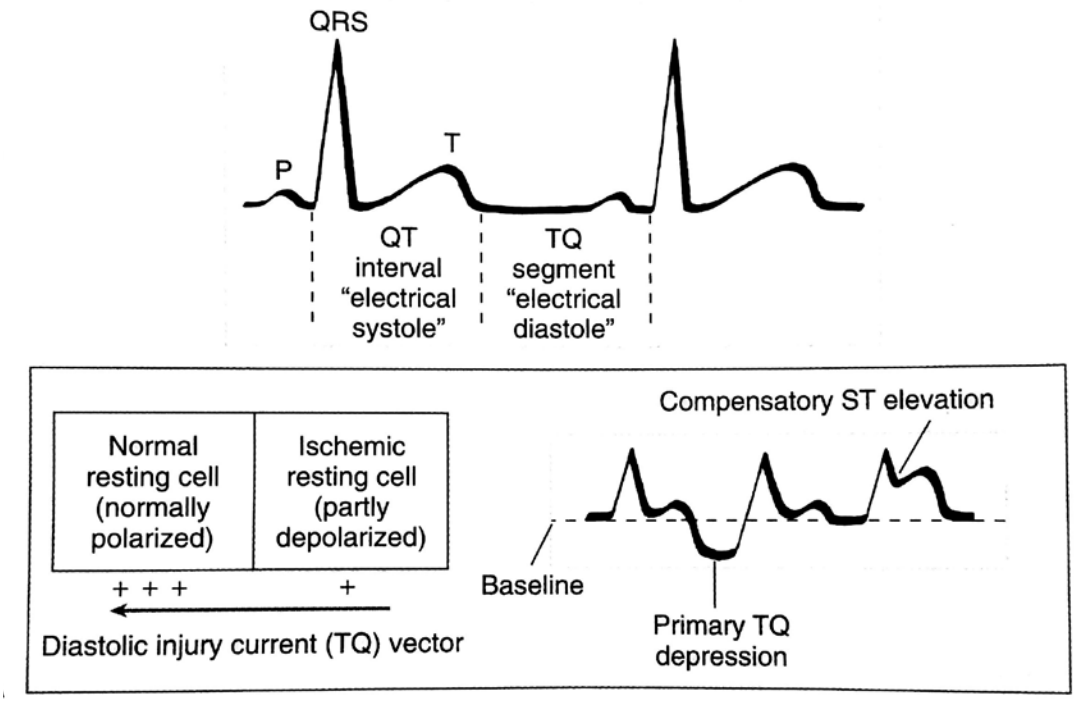
Courtesy: Braunwald's heart disease – A textbook of Cardiovascular Medicine. Chapter 13, Page 150



Two basic mechanisms are proposed to explain the ST segment elevation seen with acute myocardial injury. (32)

According to the diastolic current of injury hypothesis, ST-segment elevation is due to the negative or downward displacement of the electrical diastolic baseline (the TQ segment of the ECG).(27, 28) Ischemic myocardial cells remain relatively depolarized during phase 4 of the ventricular action potential (has lower resting membrane potential). These depolarized cells carry a negative extracellular charge relative to the repolarized myocardial cells. Thus, during electrical diastole, current flows between the partly or completely depolarized ischemic zone and the neighboring, normally repolarized, uninjured myocardium. The vector of this injury current is directed away from the more negative ischemic zone towards the more positive normal myocardium. Thus, the leads overlying an ischemic zone will record a negative deflection during electrical diastole and produce TQ-segment depression. This, in turn, appears as ST-segment elevation because ECG recorders use AC-coupled amplifiers that automatically compensate for the negative TQ-segment. As a result, the ST-segment is proportionately elevated. So the diastolic current of injury explains ST-segment elevation as an apparent shift. The true shift, that is, the negative TQ-segment is observable only with DC-coupled ECG amplifiers (Figure 3.3).(31)

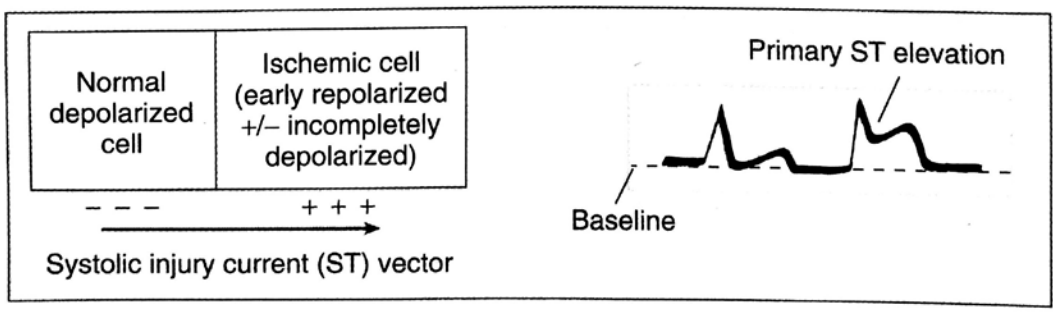
FIGURE 3.3 PATHOPHYSIOLOGY OF ISCHEMIC ST ELEVATION – DIASTOLIC INJURY CURRENT



Courtesy: Braunwald's heart disease – A textbook of Cardiovascular Medicine. Chapter 13, Page 150

The second mechanism proposed is the systolic current of injury theory. According to this postulate, ischemic myocardial cells are relatively positive in comparison with the normal cells during "electrical systole". This is due to pathologic early repolarization (shortened action potential duration), decreased action potential upstroke velocity and amplitude. As a result, a voltage gradient is established between the normal and ischemic zones. The current of injury vector is directed towards the ischemic zone resulting in ST segment elevation (Figure 3.4). (31)

FIGURE 3.4 PATHOPHYSIOLOGY OF ISCHEMIC ST ELEVATION – SYSTOLIC INJURY CURRENT



Courtesy: Braunwald's heart disease – A textbook of Cardiovascular Medicine. Chapter 13, Page 150

When acute ischemia is transmural, the overall ST vector is shifted in the direction of the epicardial layers, producing ST-segment elevation and sometimes hyperacute T waves over the ischemic zone.(32) Reciprocal ST segment depression can appear in the ECG leads representing the contra-lateral surface of the heart and can occasionally be more prominent than the primary ST-segment elevation. During sub-endocardial ischemia, the overall ST vector shifts towards the endocardium and ventricular cavity. This produces ST-segment depression in the anterior precordial leads and ST-segment elevation in lead aVR which is the typical finding during spontaneous episodes of angina, and symptomatic or silent ischemia induced by exercise or the various pharmacologic stress tests.

OTHER ISCHEMIC ST –T PATTERNS/ U WAVE CHANGES

Coronary vasospasm can cause very transient ST-segment elevation which can resolve completely within minutes or may be followed by T wave inversion that can persist for hours to days. (31)

Some patients develop deep T wave inversion in multiple precordial leads. This is typically found in high-grade stenosis in the proximal LAD (referred to as the LAD-T wave or Wellens T-wave). This T wave inversion may be preceded by a transient ST-segment elevation.

Some patients with baseline ECG abnormality can have paradoxical T wave normalization (pseudo normalization) during episodes of acute ischemia.

Alterations in U wave amplitude or polarity can be seen with acute ischemia or infarction.(34) Rarely, U wave inversion may be the earliest ECG sign of an acute coronary syndrome.

EFFECT OF STEMI ON QT INTERVAL

The QT interval is measured from the onset of the QRS complex to the end of the T wave on the surface ECG. QT interval consists of the total duration of ventricular activation and recovery, that is, ventricular APD. Accurately measuring the QT interval is challenging for various reasons, including identifying the beginning of the QRS complex and end of the T wave, determining which lead(s) to use, adjusting the measured interval for rate, QRS duration and gender(35). The duration of the QT interval varies widely in the general population(29). The normal QT interval decreases as the heart rate increases, as does the duration of the normal ventricular action potential duration and refractoriness. Thus, the normal range for the QT interval is rate-dependent. Numerous formulas have been proposed to correct the measured QT interval for this rate effect. A commonly used formula was developed by Bazett in 1920.

Corrected QT interval (QTc) = $QT/\sqrt{RR}$, where the QT and RR intervals are measured in seconds. The normal QTc is < 440 ms and is slightly longer in women <40 years.

The difference between the minimum and maximum QT interval in the same 12-lead surface ECG is the QT dispersion. Normally, it can be up to 50 to 65 ms.(36) CQTd is representative of the regional variations in the excitability and recovery of the ischemic myocardium.

Ischemia results in abrupt reduction in the trans-membrane resting potential (RMP), reduced upstroke velocity, amplitude and duration of the action potential.(37) When ischemic cells depolarize to RMP less than -60 mV, they may become inexcitable and the refractoriness of this

tissue increases. The consequence is islands of slow conduction and electro-physiologic heterogeneity which creates unidirectional block and the substrate for reentry.(37) This dispersion of refractory periods produced by acute ischaemia manifest as increased QT dispersion which is further enhanced by a healed ischemic injury and forms the substrate for reentrant tachycardias and VF.(38) The time course of repolarization is lengthened after healing of ischemic injury and shortened by acute ischemia.

Different studies have shown that CQTd increases acutely after STEMI.(39) Reperfusion therapy restoring flow in the infarct-related artery decreases it. The increased CQTd post STEMI was due to lengthening of the maximum QTc and shortening of the minimum QTc.(40) The CQTd reduction was significant when TIMI III flow was achieved. Prolongation of the ventricular repolarization (QT interval) is related to the underlying ischaemia post STEMI.(41)

EFFECT OF STEMI ON TpTe INTERVAL

The action potential duration (APD) in the ischemic zone is affected by various metabolic and electrochemical changes. This includes alterations of tissue oxygen levels, pH, and intracellular and intercellular electrochemical gradients. There is initially a transient increase followed by shortening of the APD. This is due to reduction in transmembrane potential, APD and upstroke velocity of the action potential. All these changes in the ischemic zone are not uniform and leads to a steep dispersion of APD across the zone of ischaemia.(42) Cell-to-cell interactions are also abnormal due to closing and redistribution of gap junctions. Altogether, the dispersion between normal non-ischemic and the infarcted tissue is increased during ST elevation MI and this is a substrate for various ventricular arrhythmias. This dispersion is reflected by the increased TpTe interval. TpTe interval is easy to determine during the acute phase after STEMI for risk stratification.

The correlation of TpTe with risk of sudden cardiac death in acute coronary syndrome setting is debatable. The high negative value of TpTe is comparable to that obtained with T wave alternans and signal averaged ECG analyses.(39, 41) The normal TpTe in healthy individuals is below 100 milli-seconds.(45) In the study by Christian Haarmark et al, the pre TpTe interval was 102 ms for 91 patients pre primary PCI who survived versus 122 ms in 10 patients who did not.(46) However, there are studies where no association was found between pre TpTe interval and adverse effects. Zabel et al investigated TpTe interval in 280 patients with MI. There was no significant difference in TpTe interval between survivors and non-survivors. Similarly, no significant difference in TpTe interval was found between patients with or without arrhythmias

in the follow-up period.(47) On the contrary, in the study by Bonnemeier et al, TpTe interval assessed from Holter recordings hourly was significantly higher in MI patients who had major arrhythmias in the first 24 hours after PCI. Also, TpTe interval was shown to fall in the first 4 hours after PCI, and remained in a steady level there after.(48)

EFFECT OF REPOLARIZATION ON SUDDEN CARDIAC DEATH

Sudden cardiac death is natural death from cardiac causes, heralded by abrupt loss of consciousness within 1 hour of the onset of an acute change in cardiovascular status.(49)

The identification of specific clinical markers of risk of SCD in coronary artery disease has been a goal for many years. Coronary artery disease and its consequences account for at least 80% of SCDs in Western countries. (50)

Electrical mechanisms of cardiac arrest and SCD are divided into tachy-arrhythmic and bradyarrhythmia-asystolic events. The tachyarrhythmias include VF and pulseless or sustained VT. Bradyarrhythmia-asystolic events consist of severe bradycardias, asystole (complete absence of electrical activity) or dissociation between abnormal spontaneous electrical activity and mechanical function (pulseless electrical activity). (37)

Electrophysiological characters of the myocardium has shown promise to be able to predict malignant arrhythmias. The various parameters that can be measured are QT interval, signal averaged ECG, and T wave alternans.(43) However, all these values are not validated enough to be used in clinical trials.

Many clinical studies have shown that increased CQTd can identify an increased arrhythmogenic risk in patients with long QT syndromes, hypertrophic cardiomyopathy and heart failure.(51) However, the same has not been shown in patients with ACS.

Prolonged TpTe has been associated with increased risk of arrhythmia and sudden cardiac risk in various clinical conditions like LQTS, Brugada syndrome and CPVT. There is evidence of TpTe as an index for predicting torsades de pointes in Long QT syndrome.(52) In hypertrophic cardiomyopathy, TpTe is a predictor of sudden cardiac death, but not QT dispersion).(53) In cases of bradyarrhythmia induced torsades, TpTe was the best single predictor of torsades compared to QT and QTc. Watanabe et al showed that prolonged TpTe was associated with inducibility and spontaneous development of VT in organic heart disease.(54) However, there is a paucity of studies determining the predictive value of TpTe in coronary artery disease.

CHAPTER 4

METHODOLOGY

SETTING

Department of Cardiology in Christian Medical College, Vellore which is a 2200 bedded, tertiary care, multi-specialty teaching hospital in South India.

STUDY DESIGN

This is a prospective study done among patients diagnosed with STEMI in the Department of Cardiology in Christian Medical College, Vellore from June 2013 to December 2013.

INSTITUTIONAL REVIEW BOARD (IRB) AND ETHICS COMMITTEE APPROVAL

The institutional review board of the institution reviewed, discussed and approved the project to be conducted as presented in Appendix IV.

STUDY GROUP

Patients diagnosed with new onset STEMI who presented to the chest pain unit and were planned for reperfusion were recruited.

RECRUITMENT

This was done after the study details were explained in the language that the patient understood. The patient was provided with an information sheet and a consent form (Appendix I). Consent was taken from patients who are willing to get enrolled in the study. Proforma (Appendix II) was filled.

These patients underwent reperfusion therapy (primary PCI/ Thromobolysis). The method of reperfusion was left to the discretion of the clinician. ECGs (in 50 mm/sec speed and 20 mm/mV gain) were taken before and after reperfusion (90 minutes after thrombolysis and after the patient reached the ward after primary PCI).

INCLUSION CRITERIA

- Patients with STEMI who underwent reperfusion strategy
- Age group – 18 -80 years
- Period of recruitment: June 2013 to December 2013
- The choice of reperfusion therapy was left to the clinician.

EXCLUSION CRITERIA

Patients with STEMI who did not undergo reperfusion

- Age >80 (6 patients excluded)
- Atrial Fibrillation (2 patients excluded)
- LBBB (2 patients excluded)
- qRBBB (10 patients excluded)
- Prior MI (5 patients excluded)
- On TPI (3 patients excluded)
- When the ECG was not interpretable (18 patients excluded)

PARAMETERS RECORDED

CLINICAL VARIABLES

- Age – in years
- Sex – male or female
- Symptom at presentation – chest pain/others
- Duration of symptoms at presentation – in hours
- Risk factors
    1) Diabetes Mellitus- on Oral Hypoglycaemic Agents, Insulin or lifestyle modification
    2) Hypertension – on drugs or lifestyle modification
    3) Smoking – currently or in the last 1 year
    4) Dyslipidemia – on drugs or lifestyle modification
    5) Family history of premature CAD – males below 55 years or females below 65 years
- Killip Class at admission – class I to IV
- 30 day follow up (6 lost) –
    1) Mortality
    2) Heart failure – requiring re-admission
    3) Arrhythmias - ECG documentation

ELECTROCARDIOGRAPHIC VARIABLES

Standard 12-lead ECG were recorded at 50 mm/s paper speed and 20 mm/mV gain

- Site of infarction – anterior or inferior
- ST elevation – in millivolts
- Tpeak Tend interval (TpTe)

  TpTe were evaluated in the non-infarct leads, that is, leads with ST deviation < 0.5 mV at the J point in the pre-perfusion ECG, to avoid difficulties in assessing T wave markers. The intervals between Q onset and T-wave peak (QTp) and T wave end (QTe) were manually measured. The TpTe interval is defined as the difference between the QTe and QTp intervals. The averages of all the values were taken.

- QT Dispersion

  The QT interval was measured manually from the onset of the QRS complex to the end of the T wave, defined as the point of return of the T wave to the isoelectric line or to the nadir between the T and U waves (in cases where a U wave is present). Whenever possible, the average measurement of 3 complexes for each lead was taken. If the end of the T wave could not be determined reliably or when the T wave is isoelectric or of very low amplitude, the QT measurement was not made in that lead and was excluded from the analysis. In order to exclude the effect of heart rates on QT intervals, these were corrected using Bazett's formula. Corrected QTd is defined as the difference between maximum and minimum QTc.

ECHOCARDIOGRAPHIC VARIABLES

All the patients underwent Transthoracic Echocardiography done by Cardiology Registrars during Hospital admission. All the parameters were recorded in accordance with the guidelines from the American Society of Echocardiography. Left ventricular Ejection Fraction was measured using Simpson's method. Measures of diastolic dysfunction were assessed from 2D mode and Tissue Doppler mode using pulsed wave Doppler.

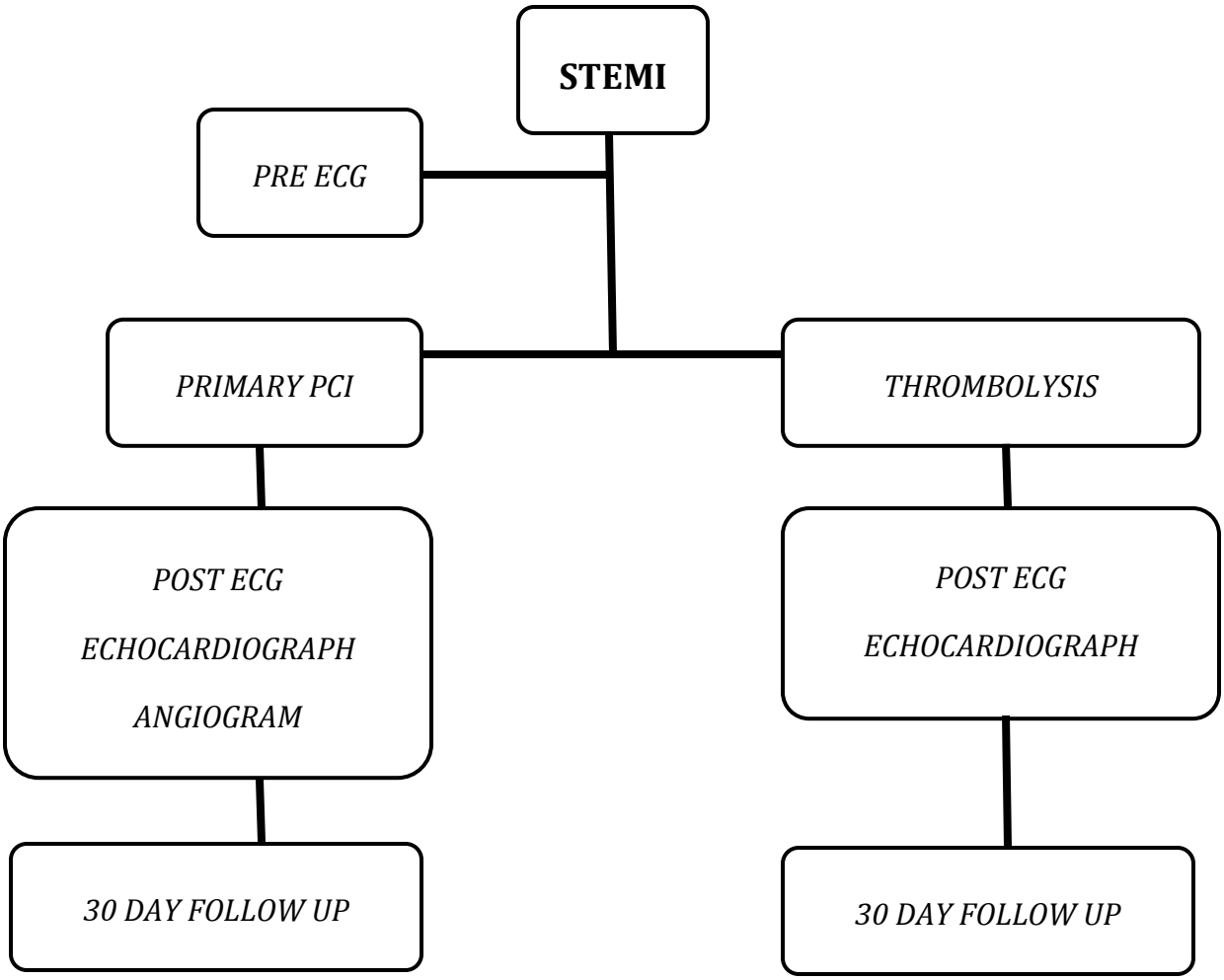
- Left ventricular Ejection Fraction (Simpson's method)
- E/A (using Pulsed wave Doppler of Mitral inflow velocity)
- Mitral deceleration Time- in milliseconds(using Pulsed wave Doppler of Mitral inflow velocity)
- IVRT – in milliseconds (using Tissue Doppler)
- Septal & lateral e' velocities (using Tissue Doppler)
- E/e'

ANGIOGRAPHIC VARIABLES

All primary and rescue PCI were done by Cardiology Consultants. All the parameters were assessed at the standard 12.5 -16 frames per second. The parameters that were assessed are

- Vessel(s) involved
- TIMI flow before and after reperfusion – grade 0 to grade 3
- TIMI myocardial perfusion grade before and after reperfusion – grade 0 to grade 3
- Stent used- Drug eluting/ Bare metal stent and size

FIGURE 3.5 ALGORITHM OF THE STUDY



STATISTICAL ANALYSIS

Data entry was done using Epidata and exported to Statistical Package for the Social Sciences (SPSS) software released in 2009, PASW Statistics for Windows, Version 18.0 Chicago: SPSS Inc. Descriptive statistics were tabulated using the SPSS software. The chi-square test was used for comparison of categorical variables. Odds ratios (OR) and confidence intervals (CI) were calculated and p value <0.05 was considered statistically significant. All reported p values are 2 sided. Continuous variable were handled with Student t test and Analysis Of Variance test (ANOVA).

SAMPLE SIZE CALCULATION

Sample size was calculated using the following formula

$$N_{pairs} = \frac{\left(z_{1-\alpha/2} + z_{1-\beta}\right)^2}{\Delta^2} + \frac{z_{1-\alpha/2}^2}{2}$$

$$\Delta = \frac{(\mu_2 - \mu_1)}{\sigma} \qquad \sigma = \frac{\sigma_1 + \sigma_2}{2}$$

This was done using the following values

Power - 80%

α-error - 5%

Pre-test means - 102 ms

Post-test mean - 106 ms

Standard deviation - 30 ms

Sided - 2

Sample size = 150

A total of 216 patients were included in the final analysis.

Values were derived from "*The prognostic value of the Tpeak-Tend interval in patients undergoing primary percutaneous coronary intervention for ST-segment elevation myocardial infarction" study by Haarmark et al* (46)

CHAPTER 5

RESULTS

DEMOGRAPHIC AND CLINICAL CHARACTERISTICS

During the study period 262 patients were enrolled. However, only 216 patients met the inclusion criteria. The rest were excluded due to the following reasons

- Age >80 (6 patients excluded)
- Atrial Fibrillation (2 patients excluded)
- LBBB (2 patients excluded)
- qRBBB (10 patients excluded)
- Prior MI (5 patients excluded)
- On TPI (3 patients excluded)
- The ECG was not interpretable (18 patients excluded)

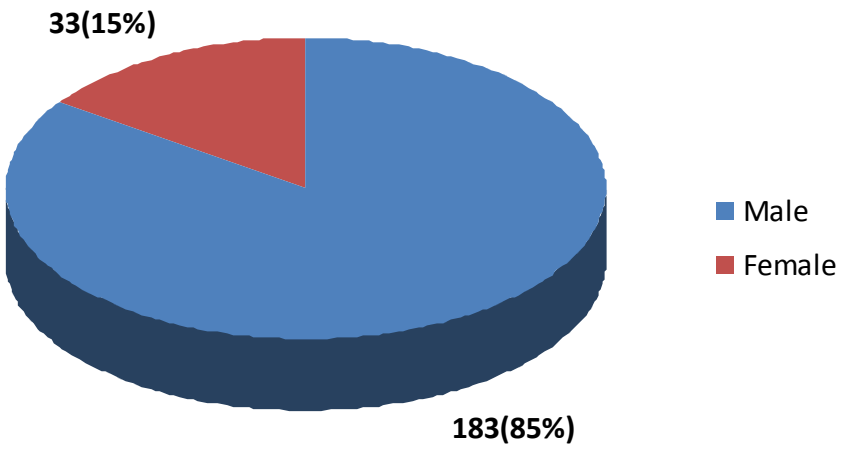
The baseline characteristics of the patients that were included are tabulated in Table 6.1

TABLE 6.1 BASELINE CHARACTERISTICS OF THE STUDY GROUP

	Male 183(84.7%)	**Female 33(15.3%)**	**Total 216**
Age(Standard Deviation)(in years)	53.4(12.0)	63.2(9.8)	54.9(12.1)
Chest pain at presentation	180(98.4%)	29(87.9%)	209(96.8%)
Duration of presenting complaint-Mean and standard deviation (in hours)	5.95(4.9)	9.88(9.7)	6.55(6.10)
PREEXISTING COMORBIDITIES			
Diabetes Mellitus	65(35.5%)	18(54.5%)	83(38.4%)
Hypertension	65(35.5%)	14(42.4%)	79(36.6%)
Smoker	100(54.6%)	0(0%)	100(46.3%)
Dyslipidemia	7(3.8%)	1(3%)	8(3.7%)
Family history of Coronary artery disease	6(3.3%)	0(0%)	6(2.8%)
KILLIP CLASS AT PRESENTATION			
I	149(81.4%)	22(66.7%)	171(79.2%)
II	21(11.5%)	8(24.2%)	29(13.4%)
III	5(2.7%)	2(6.1)	7(3.2%)
IV	8(4.4%)	1(3%)	9(4.2%)
ECG SITE OF INFARCTION			
Anterior	106(57.9%)	19(57.6%)	125(57.9%)
Inferior	77(42.1%)	14(42.4%)	91(42.1%)
INTERVENTION			
Primary PCI	95(51.9%)	18(54.5%)	113(52.3%)
Lysis	46(25.1%)	11(33.3%)	57(26.4%)
Rescue PCI	42(23%)	4(12.2%)	46(21.3%)

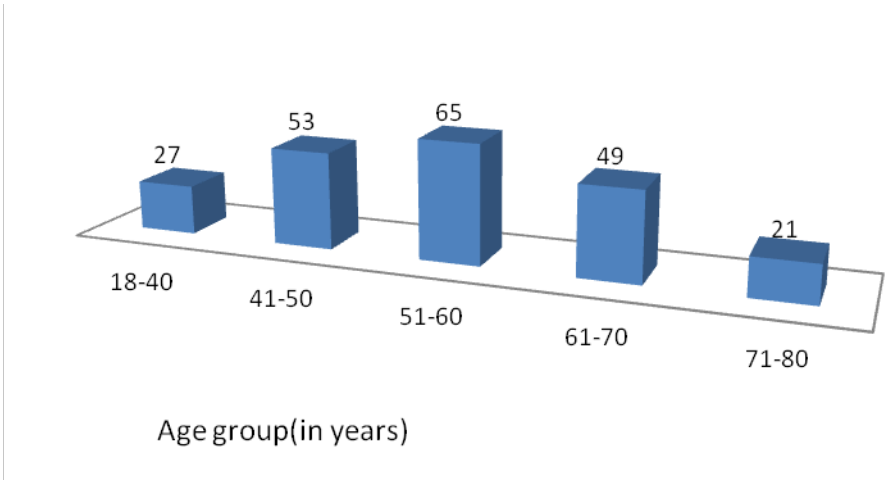
The study group included 216 patients, with 183(85%) males and 33(15%) females (Figure 6.1).

FIGURE 6.1 SEX DISTRIBUTION OF THE STUDY POPULATION



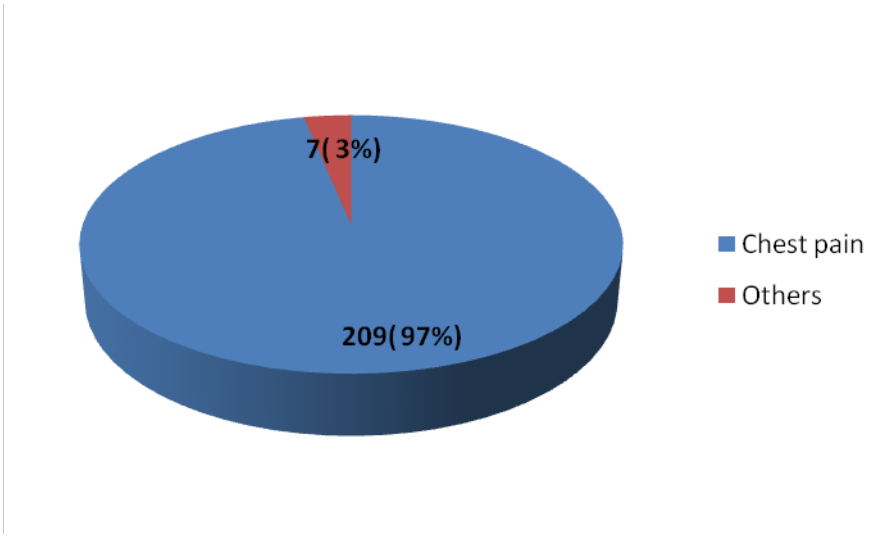
The most common age groups that presented with STEMI were in the age group of 51 – 60 years followed by 41 -50 years and 61-70 years (Figure 5). Men presented at a younger age (53.4 years) compared to women (63.2 years) (Figure 6.2).

FIGURE 6.2 AGE GROUP DISTRIBUTION IN THE STUDY POPULATION



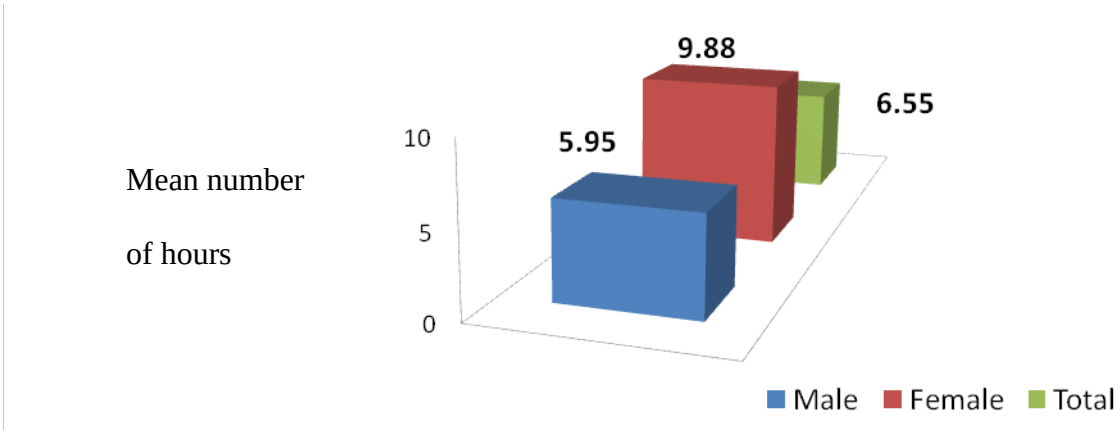
More women presented with atypical symptoms (other than chest pain) compared to men (12.1 versus 1.6%) (Figure 6.3)**.**

FIGURE 6.3 CHIEF PRESENTING COMPLAINTS IN THE STUDY POPULATION



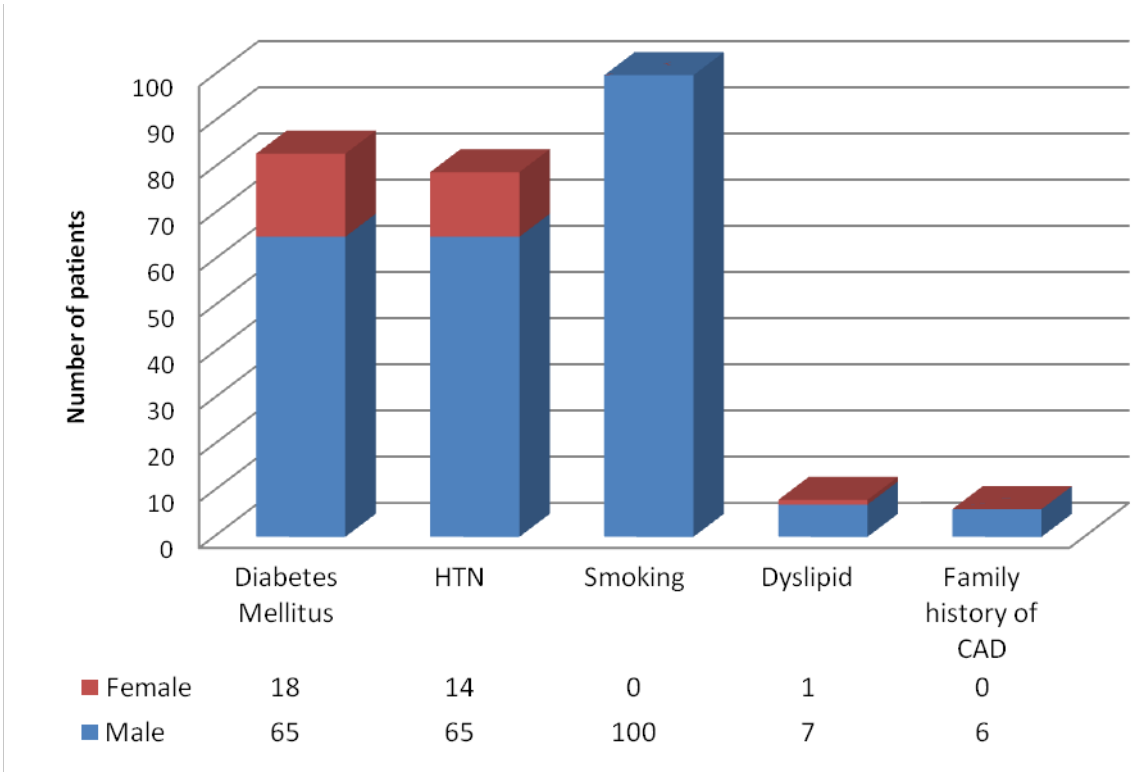
Men also presented earlier to the Hospital compared to women with STEMI (5.95 versus 9.88 hours). This finding was statistically significant with a t-test statistic value of -3.567; $p < 0.05$ (Figure 6.4)**.** Smoking was the most common risk factor for STEMI - present in 54.6% of males.

FIGURE6.4 DURATION OF SYMPTOMS AT PRESENTATION IN THE STUDY POPULATION



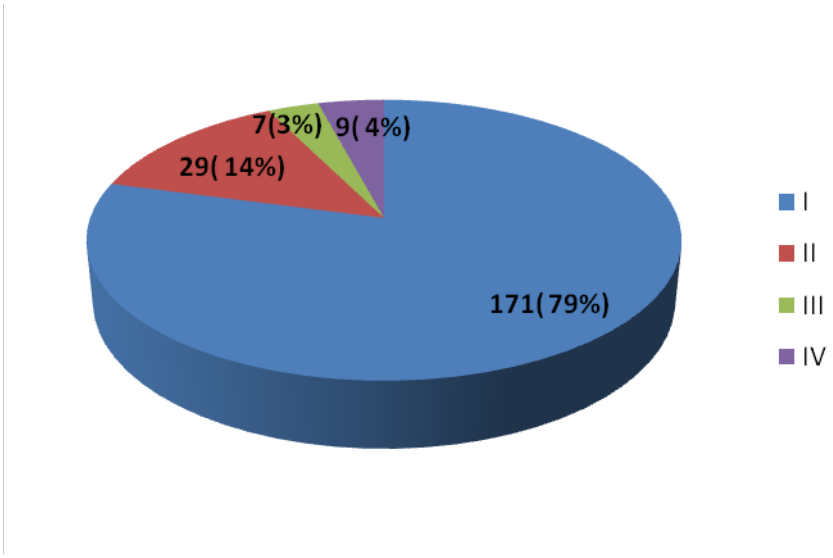
Diabetes Mellitus (men - 35.5% and women - 54.5% with p value of 0.039) and hypertension (men – 35.5% and women – 42.4% with p value of 0.448) were the next important risk factors and both of these were more prevalent among female patients. Few patients had dyslipidemia (3.7%) or family history of premature CAD (2.8%) (Figure 6.5 & Table 6.1).

FIGURE 6.5 PRE-EXISTING COMORBIDITIES IN THE STUDY POPULATION



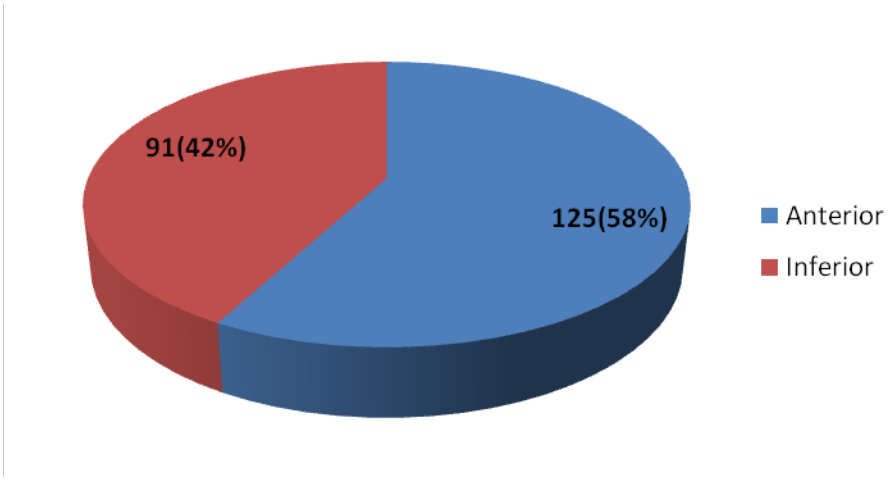
Majority of patients presented in Killip class I (79.2%). Of the rest, 7.6% of patients presented in Killip class III and IV (Figure 6.6).

FIGURE 6.6 KILLIP CLASS IN THE STUDY POPULATION



Fifty eight% of patients were found to have anterior site infarction and 42% were found to have inferior wall STEMI on ECG recordings (Figure 6.7).

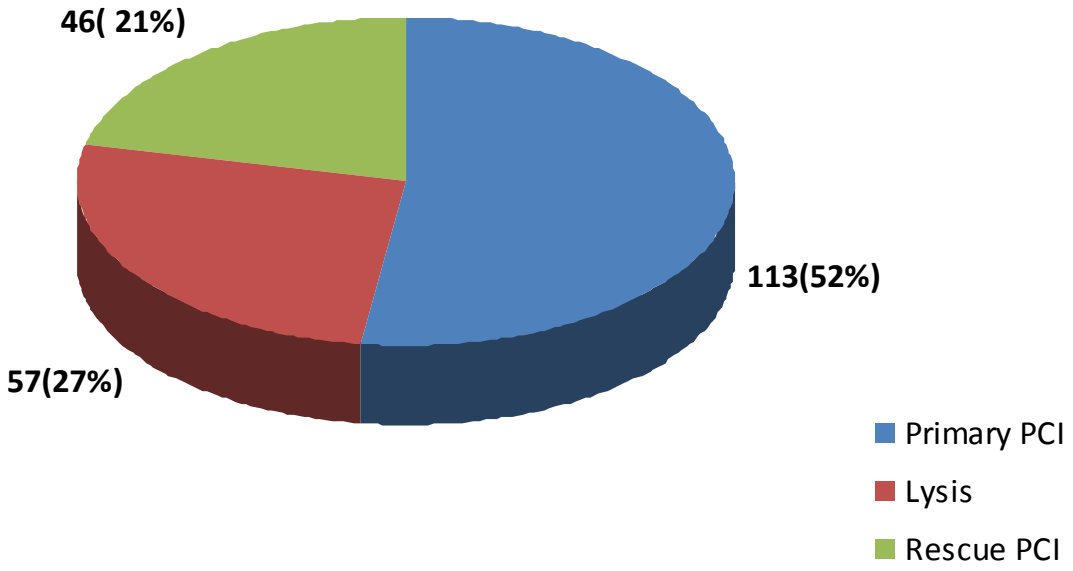
FIGURE 6.7 SITE OF INFARCTION IN THE STUDY POPULATION



REPERFUSION STRATEGY EMPLOYED

One hundred and thirteen patients underwent primary PCI, 46 had rescue PCI and 57 underwent thrombolysis with streptokinase (Figure 6.8).

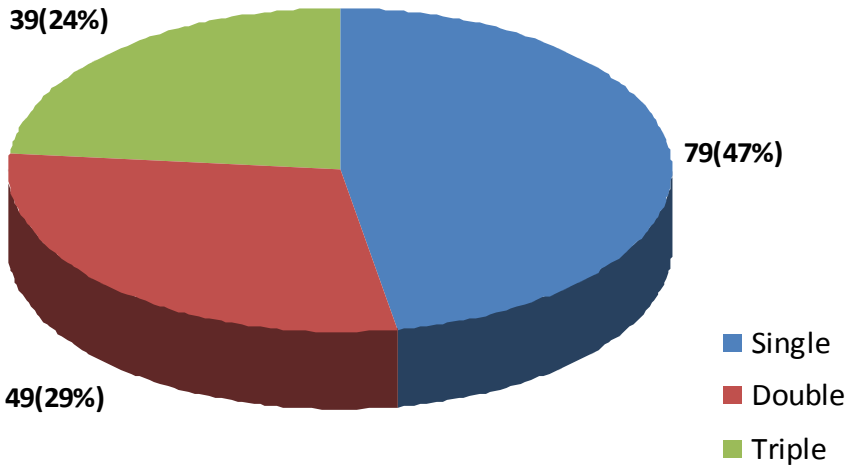
FIGURE 6.8 REPERFUSION STRATEGIES USED IN THE STUDY POPULATION



ANGIOGRAPHIC PROFILE OF THE STUDY POPULATION

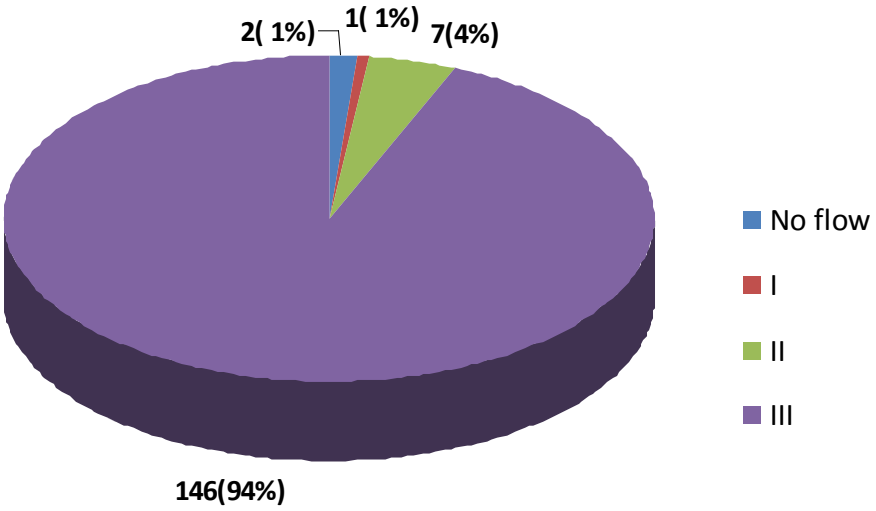
Of the 167 patients who underwent primary or rescue PCI, 79(47%), 49(29%) and 39(24%) had single, double and triple vessel coronary artery disease respectively (Figure 6.9)

FIGURE 6.9 ANGIOGRAPHIC FINDINGS IN THE STUDY POPULATION



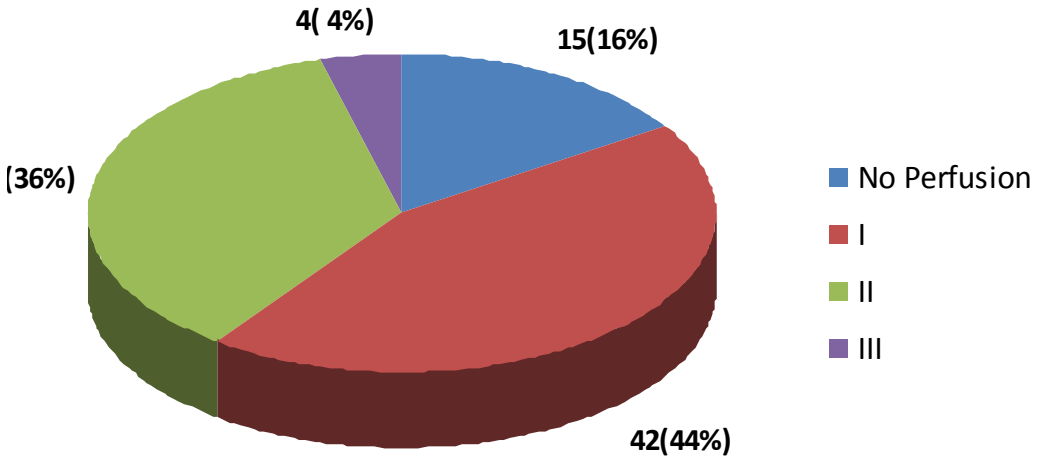
Of the 156 patients who underwent PCI 146(94%) achieved TIMI 3 flow, 7(4%) achieved TIMI 2, 1(1%) achieved TIMI 1 and 2(1%) patients had no flow (Figure 6.10).

FIGURE 6.10 CHANGES IN THE THROMBOLYSIS IN MYOCARDIAL INFARCTION (TIMI) FLOW IN THE CULPRIT ARTERY AFTER PERCUTANEOUS CORONARY INTERVENTION



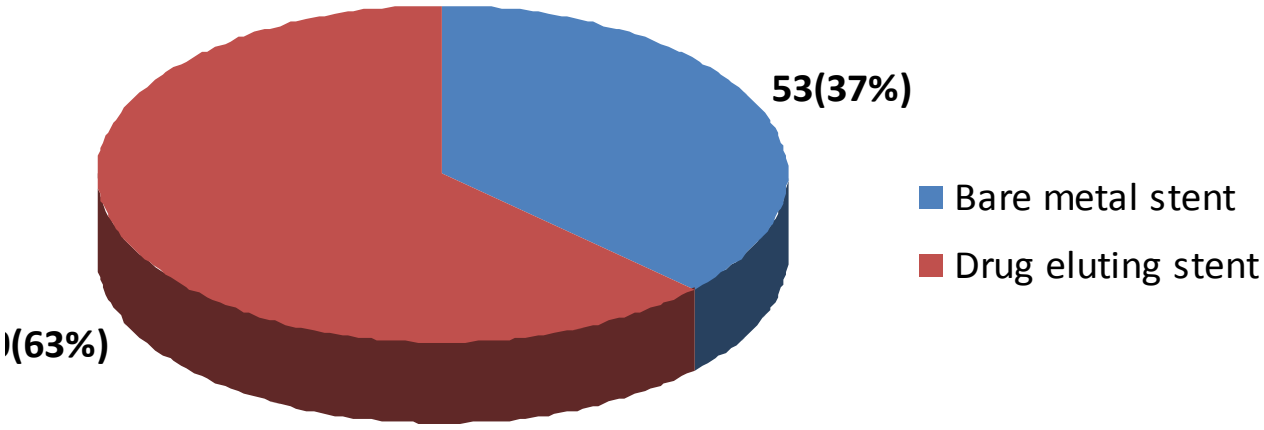
Of the 95 patients, TMP grading after PCI indicated that only 4(4%) had achieved TMP III flow, 34(36%) TMP II, 42(44%) TMP I and 15(16%) patients had no perfusion (Figure 6.11).

FIGURE 6.11 CHANGES IN THE TIMI MYOCARDIAL PERFUSION (TMP) GRADING IN THE CULPRIT ARTERY AFTER PERCUTANEOUS CORONARY INTERVENTION



Of the 143 patients who underwent PCI with stenting 90(63%) had Drug eluting stent and 53(37%) had Bare metal stent inserted (Figure 6.12).

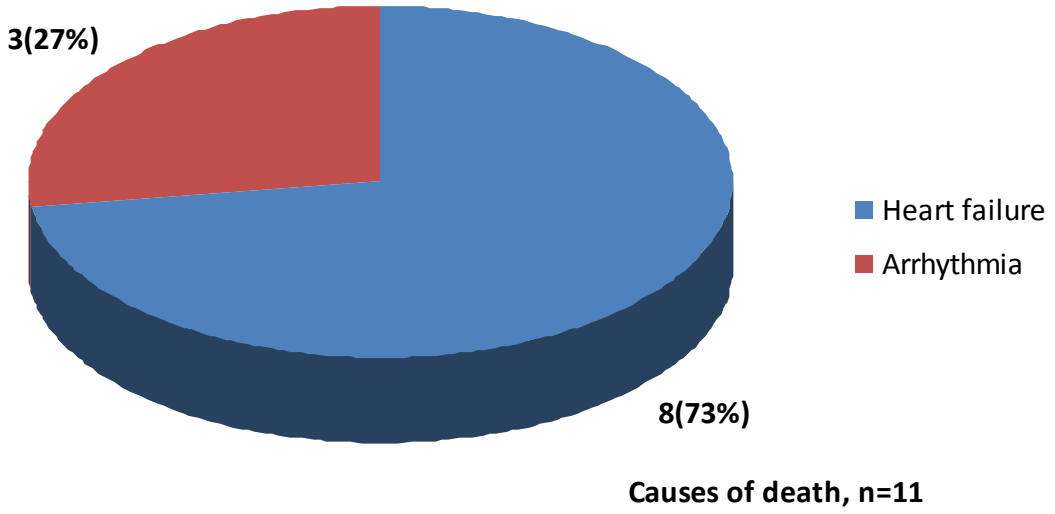
FIGURE 6.12 TYPE OF STENT USED IN PRIMARY AND RESCUE PERCUTANEOUS CORONARY INTERVENTION



MAJOR ADVERSE CARDIAC EVENTS(MACE) AT 30 DAYS

There were a total of 11(5.1%) deaths on 30 day follow up. Of these patients, 8 had heart failure and 3 had ventricular arrhythmias (Figure 6.13).

FIGURE 6.13 MAJOR ADVERSE CARDIAC EVENTS (DEATH, HEART FAILURE AND ARRHYTHMIA)AT 30 DAYS



EFFECT OF REPERFUSION ON REPOLARIZATION INDICES

EFFECT OF REPERFUSION ON TPTE INTERVAL

There was a significant reduction in duration of repolarization following reperfusion as denoted by reduction in TpTe interval in the three intervention groups (primary PCI, lysis and rescue PCI) with p values of < 0.001, 0.001 & 0.005 (Table 6.2).

TABLE 6.2 PRE AND POST TPTE CORRELATION IN THE INTERVENTION GROUPS - PAIRED SAMPLES STATISTICS

Intervention	25th percentile	50th percentile	75th percentile	Difference in pre & post TpTe	Correlation between pre & post TpTe interval(ms)	Significance level (p value)
Primary PCI N=113	80	84	100	13.56	0.544	<0.001*
Lysis N=56	80	82	98	13.55	0.534	<0.001*
Rescue PCI N=46	80	86	100	18.68	0.415	<0.005*

**p<0.05, paired T test statistic*

The median pre TpTe interval was 84.5ms and the 25th, 50th and 75th percentiles were 80, 84 and 100 ms respectively. The median post TpTe intervals were 76.7ms (64, 76.7 and 80ms), 75ms (60, 75 and 80ms) and 73.3ms (66.7, 73.3 and 80ms) respectively in the primary PCI, thrombolysis and rescue PCI groups.

The TpTe interval was reduced by all reperfusion strategies and there was no added advantage using any of the treatment options (Table 6.3).

TABLE 6.3 DIFFERENCE BETWEEN PRE POST INTERVENTION TPTE INTERVALS BETWEEN DIFFERENT TREATMENT GROUPS – ANOVA

	Sum of Squares	df	Mean Square	F	Significance level (p value)
Between Groups	947.87	2	473.93	2.31	.101
Within Groups	43419.16	212	204.80		
Total	44367.03	214			

EFFECT OF REPERFUSION ON QT DISPERSION

There was a significant increase QT dispersion in the three intervention groups (primary PCI, lysis and rescue PCI) with p values of <0.001, 0.002 & 0.009 (Tables 6.4).

TABLE 6.4 PRE AND POST CQTD CORRELATION IN THE INTERVENTION GROUPS - PAIRED SAMPLES STATISTICS

Intervention	25^{th} percentile	50^{th} percentile	75^{th} percentile	Difference in pre & post CQTd	Correlation between pre & post CQTd interval(ms)	Significance level (p value)
Primary PCI N=113	31.5	59	86.5	12.45	0.419	<0.001
Lysis N=56	19.5	46	72	11.81	0.426	<0.002
Rescue PCI N=46	25	44.5	69.75	11.94	0.387	<0.009

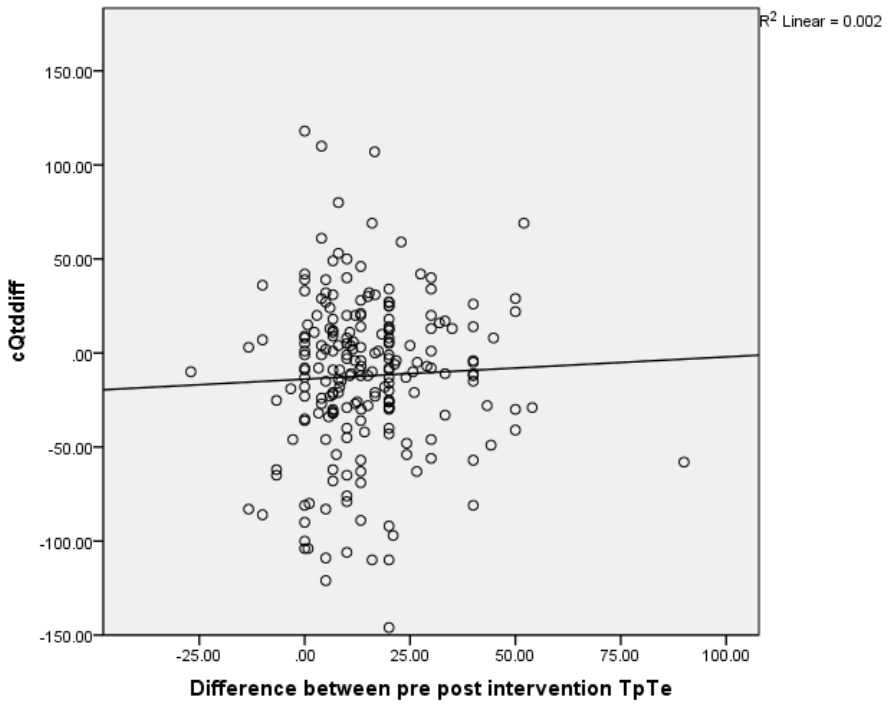
**p<0.05, paired T test statistic*

However, there was no correlation in the ΔTpTe interval and ΔCQT dispersion (Table 6.5 & Figure 6.14)

TABLE 6.5 CORRELATION BETWEEN PRE AND POST TPTE AND CQTD

	CQTd difference	
Difference between pre & post intervention TpTe interval (n=215)	Correlation coefficient Spearman's rho	Significance level (p value)
	.050	0.465

FIGURE 6.14 CORRELATION BETWEEN ΔTPTE AND Δ CQTD - NON-LINEAR GRAPH



EFFECT OF VARIOUS RISK FACTORS ON ΔTpTe INTERVAL

A multivariate linear regression analysis was performed to study the association of various risk factors with the change in TpTe intervals pre and post intervention. ΔTpTe was found to be significantly associated with duration of chest pain and Killip class at presentation (Table 6.6)

TABLE 6.6 MULTIVARIATE LINEAR REGRESSION ANALYSIS PREDICTING THE ΔTPTE AND VARIOUS RISK FACTORS

Model	B	Significance level (p value)	95.0% Confidence Interval for B	
			Lower bound	Upper bound
Constant	-2.954	.004	-36.771	-7.335
Age in years	-1.435	.153	-.261	.041
Male gender	1.281	.202	-1.819	8.565
Duration of presenting complaint(hours)	-2.620*	.009	-.663	-.094
Diabetes mellitus present	-1.229	.221	-5.836	1.355
Hypertension present	.530	.597	-2.613	4.533
Smoking present	-1.313	.191	-6.292	1.263
Dyslipidemia present	.119	.905	-8.348	9.422
Family history of premature CAD present	-.962	.337	-15.203	5.232
Killip class(I-IV)	-2.340*	.020	-5.211	-.445
Anterior infarct on ECG	.388	.698	-2.785	4.151

**Statistically significant at p <0.05*

There was no statistically significant correlation in pre TpTe interval and Left Ventricular Ejection Fraction (by Simpson's method) with p value of 0.880 (Table 6.7).

TABLE 6.7 CORRELATIONS BETWEEN PRE TPTE INTERVAL AND LEFT VENTRICULAR EJECTION FRACTION

Pre TpTe interval (n=215)	**Left Ventricular Ejection Fraction**	
	Correlation coefficient Spearman's rho	Significance level (p value)
	.010	0.880

There was no statistically significant correlation in pre TpTe interval and extent of coronary artery disease with p value of 0.733 (Table 6.8).

TABLE6.8 CORRELATION BETWEEN PRE TPTE INTERVAL AND EXTENT OF CORONARY ARTERY DISEASE - ANOVA

	Sum of Squares	**df**	**Mean Square**	**F**	**Significance level (p value)**
Between Groups	142.22	2	71.11	.311	.733
Within Groups	38441.53	168	228.81		
Total	38583.76	170			

MAJOR ADVERSE CARDIAC EVENTS (MACE) AT 30 DAYS AND ASSOCIATION WITH TpTe INTERVAL

Overall 5.1% (11/216) of patients had died by the end of 30 days. The 30 day mortality was higher among the patients with Pre TpTe interval above 100 ms (6.7% Versus. 4.8%). This however was not statistically significant (Table 6.9).

TABLE 6.9 ASSOCIATION BETWEEN PRE TPTE AND 30 DAY MORTALITY

		30 day Mortality		Total	Odds ratio	95% Confidence Interval	
		No	Yes			Lower bound	Upper bound
Upto 100 (ms)		177	9	186	1.405	.288	6.842
	% within Based on 100	95.2%	4.8%	100.0%			
101 (ms) and above		28	2	30			
	% within Based on 100	93.3%	6.7%	100.0%			
Total		205	11	216			
	% within Based on 100	94.9%	5.1%	186			

Chi square test value = 0.179; p>0.05

Of the patients who had died, 8 patients died of heart failure. The 30 day heart failure was higher among the patients with Pre TpTe interval more than 100 ms (6.7% Versus. 3.2%). This association was also not statistically significant (Table 6.10).

TABLE 6.10 ASSOCIATION BETWEEN PRE TPTE AND 30 DAY HEART FAILURE

		30 day heart failure		Total	Odds ratio	95% Confidence Interval	
		No	Yes			Lower bound	Upper bound
Upto 100 (ms)		180	6	186	2.143	.412	11.148
	% within Based on 100	96.8%	3.2%	100.0%			
101(ms) and above		28	2	30			
	% within Based on 100	93.3%	6.7%	100.0%			
Total		208	8	216			
	% within Based on 100	96.3%	3.7%	100.0%			

Chi square test value = 0.858; p>0.05

Overall 1.4% (3/216) of patients had ventricular arrhythmias diagnosed on surface ECG. All three patients died. This was higher among the patients with Pre TpTe interval above 100 ms (6.7% Versus. 0.5%) which was statistically significant with an odds ratio of 13.21 (1.16 – 150.57) (Table 6.11).

TABLE 6.11 ASSOCIATION BETWEEN PRE TPTE AND 30 DAY ARRHYTHMIA

		30 day arrhythmia		Total	Odds ratio	95% Confidence Interval	
		No	Yes			Lower bound	Upper bound
Upto 100 (ms)		185	1	186	13.214	1.160	150.571
	% within Based on 100	99.5%	.5%	100.0%			
101(ms) and above		28	2	30			
	% within Based on 100	93.3%	6.7%	100.0%			
Total		213	3	216			
	% within Based on 100	98.6%	1.4%	100.0%			

Chi square test value = 7.086; p>0.05

CHAPTER6

DISCUSSION

BASELINE DEMOGRAPHICS

In this study, of the 216 patients enrolled, 183 were men (84.7%). The mean age of presentation was 10 years lesser for men compared to women (53.4 versus 63.2 years). This was comparable to the longitudinal studies from India. The mean age of the 1459 patients in a study by Kunwar et al assessing the reperfusion trends in STEMI was 56.3±11.8 years and that of the CREATE registry of acute coronary syndrome was 57.5±12 years(59, 60)

The average time to admission after symptom onset was 6.5 hours which was almost comparable to observations of the CREATE registry where the average time was 5 hours and but lagged far behind the 1.7 hours of NCDR AR-G registry.(55–57) This may be attributed to efficient ambulance service and awareness in the developed countries whereas such a system is still in it's infancy in rural Vellore. We also found that men presented earlier to the hospital after symptom onset (5.9 hours among men and 9.9 hours among women). This is comparable to what was found by Moser DK et al and may explain worse prognosis in women who suffer STEMI. (58)

Among risk factors, smoking was the most common (54.6% of men were smokers). Women had higher prevalence of Diabetes and Hypertension compared to men. This observation is

comparable to what was found in the INTERHEART study. Six people (2.8%) had a family history of premature coronary artery disease.

Almost all the men presented with chest pain (98.4%), whereas 12.1% of women had symptoms other than chest pain at presentation. Among both sexes, majority of patients presented in Killip class I (79.2% overall) which is comparable to what was observed and published by Grieco et al where it was 78.9%.(59) Anterior wall ST-elevation MI (57.9%) was more common than inferior wall ST-elevation MI (42.1%). This observation is similar to the study by Kunwar et al.(55)

INTERVENTION

One hundred and eleven patients underwent primary PCI (52.3%), followed by thrombolysis-57 patients (26.4%). Forty-six patients underwent rescue PCI (21.3%). This rate is much higher than what was done in this same institute as published by Kunwar BK et al from 2008 – 2011 where only 11% among 1905 patients underwent primary PCI.(55) The CREATE registry showed a primary PCI rate of 8%.(56) However, this is still significantly lesser than the west as described in the NCDR AR-G registry where the rate was 75.3%.(57) The recent implementation of State Health Insurance Schemes have resulted in higher rate of primary PCI in the city. This was not the case earlier where thrombolysis was the only affordable option available.(55)

TpTe INTERVAL

The ΔTpTe (pre TpTe interval -post TpTe interval) was reduced in all the intervention groups, and was statistically significant in all the groups. The median pre TpTe interval was 84.5ms and the 25^{th}, 50^{th} and 75^{th} percentiles were 80, 84 and 100ms respectively. The median post TpTe intervals were 76.7ms (64, 76.7 and 80ms), 75ms (60, 75 and 80ms) and 73.3ms (66.7, 73.3 and 80ms) respectively in the primary PCI, thrombolysis and rescue PCI groups. This was similar to the study by LIN Xiao-ming et al, where the TpTe interval was significantly reduced after PCI in patients with severe coronary artery stenosis.(60) In contrast, in the study by Christian Haarmark et al found that the, TpTe interval was prolonged after primary PCI in the survivor group but decreased in patients that died during follow up, with a hazard ratio of 10.5.(46)

The different reperfusion strategies did not confer any advantage with respect to change in TpTe interval. There was no previous study that had assessed pre and post TpTe interval in thrombolysed patients.

QT DISPERSION

The ΔCQTd (pre CQTd -post CQTd) had increased in all three intervention groups, and this increase was statistically significant in all the groups. Earlier studies however had shown that though CQTd increased in the early phase of STEMI, it decreased following reperfusion.(61–63). This differed from the observation in our study and may relate to the timing of the ECG following reperfusion. In our cohort, it was done 90 minutes after reperfusion while in the previous studies it was done after longer intervals.(64) Therefore it is plausible that the time

period following STEMI is also one of the important determinants of QT interval and its dispersion.(65) Additionally the reperfusion strategy employed also differed.

There was lack of correlation between ΔTpTe (pre TpTe interval -post TpTe interval) and ΔCQTd (pre CQTd -post CQTd). QT interval encompasses both ventricular depolarization and repolarization. Reperfusion restores action potential duration which was reduced during the ischemic state resulting in relative prolongation of the ventricular depolarization. This is manifested by a change in QRS duration on surface ECG which in turn affects the QT interval. The time line in which the changes in QT interval and TpTe interval occurs also differ. Bonnenmeier et al has shown that myocardial ischemia and reperfusion initially causes a large increase in the QT interval and later there is a decrease but it does not return to baseline values in the first 24 hours after reperfusion. The TpTe interval on the other hand was observed to decrease within the first 4 hours after PCI. (48) In this study, the post reperfusion ECG was taken within 90 minutes and hence identified the change in TpTe interval but was too early to detect change in QT interval.

When we looked at multi-variate correlation between ΔTpTe and various clinical parameters like age, gender, duration of presenting complaints, the risk factors, ECG site of infarction, Killip class, extent of coronary artery disease(single versus multi-vessel disease) ; only duration of symptom and Killip class at presentation showed significant correlation with ΔTpTe. In the study by Christian Haarmark et al also, there was no correlation between TpTe and culprit vessel or LVEF.(46) However, in the study by LIN Xiao-ming et al, TpTe interval was found to correlate with the extent and severity of coronary artery disease.(60) Prolonged duration of

presentation and higher Killip class are independent risk factors for poor prognosis after STEMI.(15) So it can be assumed that such patients with higher pre TpTe interval will benefit maximum from reperfusion therapy, preferably from primary PCI.

TpTe INTERVAL AND MAJOR ADVERSE CARDIAC EFFECTS

As the healthy population has TpTe interval <100 ms, we compared 30-day mortality, heart failure and arrhythmias between the two groups of patients with STEMI as those who had pre intervention TpTe interval upto 100 ms and those who had pre intervention TpTe interval> 100 ms.(45) Though there was a trend for increased number of deaths (OR – 1.41, 95% CI – 0.29 – 6.84) and heart failure (OR- 2.14, 95% CI – 0.41 – 11.15) in the group of patients with higher pre TpTe interval (> 100 ms), this was not statistically significant. This is in contrast with the study of Christian Haarmark et al, where the pre primary PCI TpTe interval predicted all-cause mortality. (46) However, Zabel et al assessed TpTe interval after intervention in 280 survivors of STEMI and found no significant difference in those who died.(47)

The association between pre TpTe interval of >100 ms and 30 day mortality was found to be significant in this study. However, the confidence interval of this group was too wide. Nonetheless, it predicted a 16% increased risk of arrhythmias. This was again in agreement with other studies which showed that increased TpTe interval was associated with an increase in arrhythmias in patients with coronary artery disease and post acute coronary syndrome. (54, 66-67)

CHAPTER 7

LIMITATIONS

1. The patients were not followed beyond 30 days. Hence, the conclusion derived from the study applies only to Major Adverse Cardiac Events at 30 days. Perhaps, if the cohort was followed for a longer period, the positive predictive value of the TpTe interval may have become significant.
2. The TpTe interval was assessed only up to 90 minutes post reperfusion (in patients who were lysed) or earlier (patients who under went primary or rescue PCI). Changes beyond this period have not been assessed due to time constraints.
3. The repolarization indices were calculated only in the ECG leads with non significant ST changes (not related to infarct zone). So the effect of ischemia on repolarization may not be truly representative of the actual changes.

CHAPTER 8

CONCLUSION

From this study we can conclude that Pre TpTe interval cannot be used to predict 30 day Mortality or heart failure in patients who have suffered ST-segment elevation Myocardial Infarction. However, pre TpTe interval more than 100 ms may be used to predict risk of arrhythmias.

There was statistically significant reduction of TpTe interval and an increase in CQTd in all intervention groups at 90 minutes (primary PCI, thrombolysis or rescue PCI). There was no significant association between ΔTpTe interval and ΔCQTd which suggests that one index cannot replace the other.

In this study, ΔTpTe interval was significantly associated with Killip class and duration of chest pain at presentation.

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ANNEXURE I

CONSENT AND PATIENT INFORMATION SHEET

Informed Consent form to participate in a research study

Study Title:

The prognostic value of T peak to Tend interval on the surface Electrocardiogram in patients undergoing reperfusion therapy for ST segment elevation myocardial infarction.

Study Number:

Subject's Initials: _____ Subject's Name: _____

Date of Birth / Age:_____

(i) **I confirm that I have read/it has been read to me** and understood the information sheet dated _____ for the above study and have had the opportunity to ask questions. [ ]

(ii) I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. [ ]

(iii) I understand that the Sponsor of the clinical trial, others working on the Sponsor's behalf, the Ethics Committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the trial. I agree to this

access. However, I understand that my identity will not be revealed in any information released to third parties or published. [ ]

(iv) I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s) [ ]

(v) I agree to take part in the above study. [ ]

Signature (or Thumb impression) of the Subject/Legally Acceptable Representative:_____

Date: _____/_____/_____

Signatory's Name: _____

Signature of the Investigator: _____

Date: _____/_____/_____

Study Investigator's Name: _____

Signature of the Witness: _____

Date:_____/_____/_____

Name of the Witness: _____

PATIENT INFORMATION SHEET

The prognostic value of Tpeak – Tend interval on the surface Electrocardiogram in patients undergoing reperfusion therapy for ST-segment elevation Myocardial Infarction

Dated : March, 2013

Site: Christian Medical College, Vellore

Your consent

You are invited to take part in this research project. The patient information sheet contains detailed information about the research project. Its purpose is to explain to you as openly and clearly as possible all the procedures involved in this project before you decide whether or not to take part in it.

Please read this patient information sheet carefully. Feel free to ask questions about any information in the statement. Once you understand what the project is about and if you agree to take part in it, you will be asked to sign the consent form. By signing the same, you do not alter your legal rights, but you indicate that you understand the information and that you give your consent to participate in the research project.

You will be given a copy of both the Consent form and the Patient information sheet to keep as a record.

Alternatives to Participation

It is important that you understand that your participation in this study must be voluntary. This is a research project and you do not have to be involved. If you do not want to participate your medical care will not be affected in any way.

Description of the Project

Heart attack (ST-segment elevation Myocardial Infarction) requires immediate treatment. The goal of treatment of ST-segment elevation Myocardial Infarction is mechanical reperfusion (opening up the blocked coronary artery)- either by primary percutaneous coronary intervention (angioplasty) or by pharmacological thrombolysis (medicine to dissolve the clot). The goal of reperfusion is to reduce cardiac death and morbidity. Myocardial reperfusion also alters the repolarisation (electrical characteristics) of the myocardium(heart muscle). Post heart attack patients are prone to develop ventricular arrhythmias (abnormalities in the rate and rhythm of heart) which cause sudden cardiac death. There are certain parameters in the surface 12 lead Electrocardiogram which have shown clinical promise for the prediction of death and malignant arrhythmias. They can be easily calculated without doing any extra tests. However, there has been very few studies in the setting of STEMI undergoing angioplasty, and even fewer on patients undergoing thrombolysis. The purpose of the present study is to analyze prospectively, the acute effects of reperfusion on TpTe interval(an ECG parameter) and its relationship with the clinical outcome of the patients over 30 days from the heart attack. It will also compare the effect of the two modes of reperfusion on the TpTe interval.

You are invited to participate in this study because you are undergoing a reperfusion therapy for heart attack. There will be some extra ECGs taken and some extra parameters will be looked into while doing the Echocardiography and Coronary Angiography. You will not have any added risk due to the research protocol. You will be asked to follow up after 30 days from the time of your heart attack.

Possible Benefits

The study may not directly benefit you. It will however, enable us to understand the ECG changes which may predict risk of death after heart attack. These factors may benefit other people in the future.

Possible Risks

There will be no added risks due to the research protocol.

Confidentiality and Disclosure of Information

Any information obtained in connection with this project that can identify you will remain confidential. It will only be disclosed with your permission, except as required by law. If you give us your permission by signing the consent form, we plan to publish the results of the study in a medical journal. In any publication, information will be provided in such a way that you cannot be identified.

Results of Project

Results of your individual tests will be discussed with you at the end of the procedure.

Further Information of any Problem

If you require further information or you have any problems concerning this project, you can contact Dr. Subhrangshu Dey (phone 0416-2283572)

Other Issues

If you wish to discuss aspects of the study with someone who is not directly involved, you can also contact the IRB at CMC, Vellore

Participation is Voluntary

Participation is any research project is voluntary. If you do not wish to take part, you are not obliged to. If you wish to take part and later change your mind, you are free to withdraw from the project at any stage.

Your decision whether to take part or not, or to take part and then withdraw, will not affect your routine treatment, your relationshio with those treating you or your relationship with Christian Medical College, Vellore.

Before you make your decision, you can ask any questions you have about the research project. Only sign the consent form once you have had a chance to ask your questions and have received satisfactory answers.

Before deciding whether or not to take part, you may wish to discuss the project with a relative or friend or your local health worker. Feel free to do this.

If you decide to withdraw from this project, please notify before you withdraw. This will allow us to inform you if there are any health risks linked to withdrawing.

Payment for Participation

Participation in this study is voluntary and no payment will be made

ANNEXURE II

PROFORMA

Serial No :
Name:
Hospital No:
Age:
Sex:
Phone No:

Chief Presenting complain :
Duration:
Diabetes Mellitus:
Hypertension:
Smoking:
Dyslipidemia :
Family History:

Heart Rate at admission:
BP at admission:
Killip Class at admission:

ECG
Site of infarction :
Maximum ST elevation :

Lead	I	II	III	aVR	aVL	aVF	V1	V2	V3	V4	V5	V6	Avg.
Pre TpTe													
Post TpTe													
30 Day TpTe													

	Pre	Post	30 day
cQtmax			
cQT min			
cQTd			

Echocardiogram
LV Ejection Fraction :
E/A ratio :
DT :
IVRT:
Medial E' :
E/E':
Lateral E':

E/E’ :

Intervention:

For Primary PCI group
Vessel/vessels involved:
Type of lesion:
Length of lesion:
TIMI flow before & after PCI:
TIMI Myocardial perfusion grade before & after PCI:
Stent used:
Size of stent:

30 day follow up
Alive/dead
Heart Failure
Arrhythmias

ANNEXURE III

EXPANSION OF ABBREVIATIONS

Δ : difference between pre and post
ACS : Acute coronary syndrome
BMS : Bare metal stent
CAD : coronary artery disease
Cpc : chief presenting complaint
cQT : corrected QT interval
DES : Drug eluting stent
DM : Diabetes mellitus
Dyl : Dyslipidemia
Fh : Family history
HF : Heart failure
HTN : Hypertension
Inv : Intervention
Klp : killip class
LVEF : Left Ventricular Ejection Fraction
PCI : Percutaneous coronary intervention
PCI : Percutaneous transluminal coronary angioplasty
QTd : QT dispersion
Smk : Smoker
STEMI : ST-segment elevation Myocardial infarction
STR : ST-segment resolution
TDR : Transmural dispersion of repolarization
TIMI : Thrombolysis in Myocardial Infarction
TMP : TIMI myocardial perfusion
TpTe : Tpeak to T end interval

ANNEXURE IV

MASTER CHART

Master chart table headings

id – identification number
age – age of the patient
sex – gender of the patient (male -1, female-2)
cpc – chief presenting comlplaint (1 – chest pain, 2 – others)
duration – duration of presenting complaint in hours
dur12hrs – duration of presenting complaint in hours (>12 hours – 1, < 12hours – 2)
dm - Did the patient have Diabetes Mellitus? (Yes – 1, No -2)
htn- Did the patient have Hypertension? (Yes – 1, No -2)
smk – Was the patient a smoker? (Yes – 1, No -2)
dyl - Did the patient have Dyslipidemia? (Yes – 1, No -2)
fh - Did the patient have family history of premature coronary artery disease? (Yes – 1, No -2)
klp – Killip class at presentation
ecg – Electrocardiographic site of infarction (anterior -1, inferior – 2)
stemax – Maximum ST segment elevation in mV
pretpte – Pre intervention TpTe interval in ms
posttpte – Post intervention TpTe interval in ms
precqtd - Pre intervention cQTd interval in ms
postcqtd - Post intervention cQTd interval in ms
lvef – Left Ventricular Ejection Fraction%
inv – Intervention done (Primary PCI – 1, Lysis -2, Rescue PCI -3)
ves –Extent of Coronary artery disease (Single -1, Double -2, Triple -3)
pretimi - Pre intervention TIMI flow (No flow -0, penetration -1, partial flow -2, normal flow -3)
posttimi - Post intervention TIMI flow (No flow -0, penetration -1, partial flow -2, normal flow -3)
pretmp – Pre intervention TMP grading (No perfusion – 0, minimal perfusion – 1, Moderate perfusion – 2, normal perfusion-3)
posttmp – Post intervention TMP grading (No perfusion – 0, minimal perfusion – 1, Moderate perfusion – 2, normal perfusion-3)
stent – type of stent used (None -0, BMS -1, DES -2)
sized –Diameter of stent in mm
sizel – length of stent in mm
n30dm – 30 day mortality (Alive -1, Death -2)
n30dhf – 30 day heart failure (Yes- 1, No -2)
n30dary - 30 day arrhythmia (Yes- 1, No -2)
TpTediff – difference between pre & post intervention TpTe
cQTddiff - difference between pre & post intervention cQTd

ANNEXURE VI

MASTER CHART

id	age	sex	cpc	duration	dur12hrs	dm	htn	smk	dyl	fh	klp	ecg	site	stemax	pretpte	posttpte	precqtd
1	56	1	1	5	2	1	2	2	2	0	I	2	2	0.3	80	50	114
2	45	1	1	2	2	2	2	2	2	0	I	1	1	0.2	100	72.5	74
3	80	1	1	10	2	1	2	2	2	0	I	1	1	1.1	100	66.7	66
4	67	1	1	2	2	1	2	2	2	0	I	2	2	0.3	80	40	66
5	60	1	1	5	2	2	2	1	2	0	I	2	2	0.6	103.3	74.3	43
6	62	1	1	11	2	2	2	2	2	0	I	1	1	1	84	80	35
7	50	2	1	8	2	2	2	2	2	0	III	1	1	1	100	76	27
8	60	1	1	11	2	2	2	1	2	0	I	2	2	0.6	80	75	23
9	66	1	1	4	2	2	1	2	2	0	I	2	2		100	70	12
10	53	1	1	7	2	1	2	2	2	0	I	2	2	0.3	120	90	33
11	39	1	1	24	1	1	1	2	2	1	I	1	1	1	100	100	113
12	38	1	1	3	2	2	2	2	2	0	I	2	2	0.3	100	80	35
13	67	1	1	3	2	1	2	1	2	0	I	2	2	0.2	86.7	80	59
14	62	1	1	2	2	2	1	2	2	0	III	1	1	0.2	86.7	100	33
15	52	1	1	19	1	2	2	2	2	0	I	1	1		120	70	46
16	49	1	1	4	2	2	2	1	2	0	I	1	1	1.9	80	80	21
17	47	1	1	7	2	2	2	2	2	0	I	2	2	0.4	113.3	70	16
18	62	1	1	2	2	2	1	1	2	0	I	2	2	0.4	100	60	74
19	71	1	1	1	2	2	2	2	1	0	I	2	2	0.8	93.3	72	28
20	47	1	1	8	2	2	2	1	2	0	III	1	1	0.3	80	82.8	4
21	42	1	1	2	2	2	2	2	2	0	I	1	1	0.6	90	73.3	57
22	49	1	1	2	2	1	2	1	2	0	I	1	1	0.4	80	72	64
23	69	2	1	8	2	1	2	2	2	0	I	2	2	0.4	80	70	56
24	30	1	1	2	2	2	2	2	2	0	I	1	1	1	80	66.7	41
25	70	1	1	13	1	2	2	2	2	0	IV	2	2	0.5	80	86.7	197.8
26	44	1	1	5	2	2	2	1	2	0	I	2	2	0.2	76	103	44
27	56	1	1	8	2	1	1	2	2	0	II	1	1	0.5	80	80	32
28	57	1	1	7	2	1	2	2	2	0	II	1	1	0.3	80	80	26
29	47	1	1	9	2	2	1	2	2	0	I	1	1	0.4	71.4	60	11
30	52	1	1	19	1	2	2	1	2	0	I	1	1	0.8	100	93.3	64
31	56	1	1	1	2	1	1	1	2	0	I	2	2	0.6	86.7	73.3	16
32	42	1	1	3	2	2	2	1	2	0	I	1	1	0.5	94	40	80
33	42	1	1	4	2	2	2	2	2	0	I	1	1	0.9	87.5	70	66
34	74	1	1	12	1	2	2	1	2	0	I	1	1	0.3	103.3	91.4	10
35	66	2	1	5	2	2	2	2	2	0	I	1	1	0.5	60	50	98
36	67	1	1	6	2	1	1	2	2	0	I	2	2	0.3	113.3	93.3	43
37	32	1	1	4	2	2	2	2	2	0	I	1	1	1.9	120	70	89
38	69	2	1	5	2	2	1	2	2	0	II	1	1	0.5	86.7	86	115
39	28	1	1	5	2	2	2	1	2	0	I	1	1	3.2	100	68	50
40	49	1	1	6	2	1	2	1	2	0	I	1	1	1.9	110	90	28
41	40	1	1	7	2	1	2	1	2	0	I	1	1	0.8	73.3	66.7	64
42	61	1	1	5	2	2	2	1	2	0	I	2	2	0.4	66.7	70	29

ANNEXURE VI

MASTER CHART

id	age	sex	cpc	duration	dur12hrs	dm	htn	smk	dyl	fh	klp	ecg	site	stemax	pretpte	posttpte	precqtd
43	41	1	1	10	2	1	1	1	2	0	I	2	2	0.9	100	80	75
44	50	1	1	3	2	2	2	1	2	0	I	1	1	1.1	80	80	60
45	64	1	1	4	2	1	1	1	2	0	I	1	1	0.9	90	84	100
46	60	1	1	4	2	1	2	1	2	0	III	2	2	0.4	133.3	140	25
47	51	1	1	6	2	1	1	2	2	0	I	1	1	1.9	84	65	72
48	67	2	1	48	1	2	2	2	2	0	I	2	2	0.2	80	80	60
49	38	1	1	2	2	2	2	1	2	1	I	1	1	1.5	70	60	50
50	66	1	1	2	2	1	2	1	2	0	I	1	1	2.5	95	84	14
51	76	1	1	4	2	2	1	2	2	0	I	1	1	1	90	60	72
52	49	1	1	5	2	2	2	2	2	1	I	1	1	3.6	96	84	120
53	66	1	1	5	2	1	1	2	2	0	I	1	1	0.4	86.7	86.7	14
54	55	2	1	9	2	1	2	2	2	0	I	2	2	0.4	90	60	40
55	24	1	1	7	2	2	2	2	2	0	I	2	2	0.2	100	94.3	34
56	59	2	1	19	1	2	2	2	2	0	I	1	1	0.8	100	80	133
57	74	1	1	4	2	2	1	1	2	0	IV	2	2	0.4	90	40	109
58	54	1	1	6	2	2	1	1	2	0	I	2	2	1.2	100	80	18
59	44	1	1	1	2	2	2	1	2	0	I	2	2	1.1	106.7	82.5	16
60	50	1	1	4	2	2	2	1	2	0	I	1	1	1.2	120	100	10
61	61	1	1	4	2	2	2	1	2	0	I	2	2	0.4	100	100	90
62	73	1	1	4	2	2	1	2	2	0	I	2	2	2.1	80	60	50
63	33	1	1	3	2	2	2	1	2	0	I	2	2	0.6	90	88.9	12
64	47	1	1	2	2	2	2	1	2	0	II	1	1	1	120	80	130
65	38	1	1	3	2	2	2	1	2	0	I	1	1	0.7	84	73.3	62
66	62	1	2	3	2	1	2	2	2	0	IV	1	1		120		156
67	58	1	1	1	2	1	2	2	2	0	I	2	2	0.9	80	60	40
68	66	2	1	5	2	2	2	2	2	0	II	1	1	0.3	90	60	110
69	51	1	1	4	2	2	2	1	2	0	II	1	1	0.3	70	60	110
70	59	1	1	2	2	2	2	1	2	0	I	2	2	0.6	90	70	43
71	37	1	1	1	2	1	2	1	2	0	I	2	2	0.8	113.3	68.5	11
72	70	2	1	3	2	2	2	2	2	0	I	2	2	0.6	110	65.8	69
73	57	1	1	4	2	1	1	2	1	0	I	1	1	0.5	80	72	94
74	47	1	1	1	2	2	1	1	2	0	I	1	1	0.5	100	92.5	1
75	45	1	1	4	2	2	2	1	2	0	I	2	2	0.7	80	80	67
76	66	1	1	6	2	2	2	1	2	1	I	1	1	1.3	96	80	115
77	51	1	1	4	2	1	1	1	1	0	I	2	2	0.3	120	85	80
78	70	2	1	15	1	2	2	2	2	0	II	1	1	1.2	80	90	37
79	58	1	1	2	2	2	1	1	2	0	I	2	2	1.5	80	80	13
80	45	1	1	1	2	2	2	2	2	0	III	1	1	0.4	100	80	41
81	38	1	1	6	2	2	2	1	2	0	I	1	1	0.5	80	71.4	90
82	50	1	1	2	2	2	2	2	2	0	IV	1	1		80	73.3	75
83	60	1	1	8	2	2	1	1	2	0	I	1	1	1.3	80	40	10
84	80	1	1	2	2	1	1	2	2	0	I	1	1	2.5	80	66.7	46

ANNEXURE VI

MASTER CHART

id	age	sex	cpc	duration	dur12hrs	dm	htn	smk	dyl	fh	klp	ecg	site	stemax	pretpte	posttpte	precqtd
85	39	1	1	3	2	1	1	1	2	0	I	1	1	0.3	75	60	45
86	41	1	1	6	2	1	2	1	2	0	I	2	2	0.3	80	93.3	15
87	60	2	1	4	2	1	2	2	2	0	I	1	1	0.9	80	60	34
88	61	1	1	4	2	1	1	1	1	1	I	1	1	0.6	93.3	80	28
89	80	2	1	7	2	1	2	2	2	0	I	2	2	0.6	100	100	30
90	61	1	1	5	2	1	1	1	2	0	I	2	2	0.4	113.3	100	19
91	35	1	1	2	2	2	2	1	2	0	II	1	1	2.3	75	60	46
92	64	1	1	5	2	1	1	1	2	0	I	1	1	2	140	50	11
93	52	1	1	7	2	1	2	1	2	0	I	1	1	0.2	97.5	83.3	11
94	57	1	1	2	2	2	2	1	2	0	IV	2	2	0.4	120	80	42
95	50	1	1	5	2	1	2	2	2	0	I	1	1	1.2	100	84	12
96	43	1	1	3	2	2	1	2	2	0	I	2	2	0.4	93.3	66.7	10
97	58	1	1	5	2	2	1	1	2	0	I	1	1	1.5	105	100	28
98	80	2	1	2	2	1	1	2	1	0	I	1	1	1	110	93.3	40
99	63	1	1	14	1	1	2	2	2	0	I	1	1	1.1	88	80	33
100	55	1	1	3	2	2	1	2	2	0	I	1	1	1.6	80	63.3	57
101	50	1	2	10	2	2	2	2	2	0	I	2	2	0.5	72	56.7	60
102	50	1	1	19	1	1	2	2	2	0	I	2	2	0.2	96.7	80	27
103	43	1	1	6	2	2	2	2	2	0	I	1	1	0.2	86.7	80	68
104	46	1	1	5	2	2	1	1	2	0	I	1	1	1.2	66.7	60	16
105	70	1	1	8	2	2	2	1	2	0	I	2	2	0.3	85	76.7	46
106	32	1	1	1	2	2	2	1	2	0	I	2	2	0.9	86.7	65	72
107	47	1	1	3	2	1	1	2	2	0	I	1	1	0.7	90	65.8	32
108	50	1	1	3	2	1	1	2	2	0	I	1	1	0.5	80	80	22
109	47	1	1	7	2	2	1	1	2	0	I	2	2	0.9	90	84	62
110	31	1	1	1	2	2	2	1	2	0	I	1	1	5	80	68.5	72
111	49	1	1	6	2	2	2	2	2	0	I	2	2	0.2	80	77.1	60
112	53	1	1	1	2	2	1	1	2	0	I	1	1	1.8	80	75	30
113	65	1	1	3	2	1	2	2	2	0	I	2	2	0.8	80	66.7	67
114	48	1	1	4	2	2	2	1	2	0	I	2	2	0.2	80	80	12
115	53	1	1	5	2	2	2	2	1	0	I	1	1		80	54.3	66
116	45	1	1	14	1	2	2	2	2	0	I	1	1	0.3	64	53.3	55
117	48	1	1	3	2	2	2	1	2	0	II	1	1	1.5	80	60	56
118	58	1	1	1	2	2	2	1	2	0	I	2	2	0.7	73.3	60	52
119	38	1	1	8	2	1	2	1	2	0	I	1	1	0.5	73.3	55	90
120	76	1	1	2	2	1	2	2	2	0	I	2	2	0.3	70	60	20
121	45	1	1	11	2	1	2	1	1	0	I	2	2	0.3	62.5	50	49
122	71	1	1	5	2	2	2	2	2	0	I	1	1	0.5	100	88	21
123	57	1	1	4	2	1	2	1	2	0	I	2	2	0.6	93.3	80	15
124	43	1	1	7	2	1	1	1	2	0	I	2	2	0.6	80	55	15
125	38	1	1	13	1	2	2	1	2	0	II	1	1	0.5	90	64	96
126	67	1	1	2	2	2	2	1	2	0	I	2	2	0.8	80	64	50

ANNEXURE VI

MASTER CHART

id	age	sex	cpc	duration	dur12hrs	dm	htn	smk	dyl	fh	klp	ecg	site	stemax	pretpte	posttpte	precqtd
127	57	1	1	5	2	2	1	1	2	0	II	2	2	0.5	66.7	53.3	54
128	66	1	1	3	2	2	2	1	2	0	II	1	1	1.2	120	100	79
129	33	1	1	6	2	2	2	2	2	0	I	1	1	0.2	68	57.1	58
130	65	1	1	3	2	2	2	1	2	0	I	2	2	0.2	60	55	23
131	59	1	1	22	1	2	1	2	2	0	I	1	1	0.8	66.7	60	118
132	50	2	1	2	2	1	2	2	2	0	I	2	2	0.2	100	48	86
133	51	1	1	3	2	1	2	2	2	0	I	1	1	0.6	68	55	39
134	63	1	1	4	2	2	2	1	2	0	I	2	2	0.6	60	55	83
135	60	1	1	5	2	2	2	1	2	0	II	1	1	0.3	80	73.3	66
136	53	1	1	1	2	2	1	2	2	0	I	1	1	0.8	86.7	46.7	50
137	52	1	1	4	2	1	1	2	2	0	I	2	2	0.4	100	80	84
138	76	2	1	2	2	2	2	2	2	0	I	1	1	0.4	66.7	40	104
139	50	2	1	11	2	1	2	2	2	0	I	1	1	0.4	70	66.7	80
140	65	2	1	4	2	2	1	2	2	0	I	2	2	0.2	70	64	89
141	62	1	1	3	2	1	2	1	2	0	I	1	1	0.8	93.3	80	59
142	63	1	2	2	2	2	2	1	2	0	I	2	2	0.3	80	66.7	20
143	65	2	2	7	2	1	1	2	2	0	II	1	1	1	65	60	102
144	55	2	1	12	1	1	2	2	2	0	II	1	1	1.1	75	70	46
145	51	1	1	12	1	1	1	2	2	0	I	1	1	1	63.3	55	26
146	65	1	1	4	2	2	2	2	2	0	II	1	1	0.6	82.28	80	87
147	52	1	1	5	2	2	2	1	2	0	I	1	1	0.6	80	73.3	96
148	40	1	1	12	1	2	1	1	2	0	I	2	2	0.3	86.7	80	50
149	49	1	1	4	2	2	2	1	2	0	I	1	1	0.9	80	72	13
150	72	1	1	6	2	2	1	1	2	0	I	1	1	1.2	80	80	82
151	57	1	1	22	1	1	1	2	2	0	I	2	2	0.3	80	75	2
152	40	1	1	5	2	2	2	2	2	0	I	1	1	0.2	80	66.7	5
153	46	1	1	9	2	1	2	2	2	0	I	1	1	0.9	80	80	38
154	65	1	1	1	2	2	1	1	1	0	II	2	2	0.8	80	86.7	4
155	24	1	1	2	2	2	2	1	2	0	II	1	1		100	60	156
156	52	1	1	15	1	2	1	1	2	0	I	1	1		110	80	26
157	53	1	1	2	2	2	2	1	2	0	I	1	1	2.1	110	100	42
158	52	1	1	14	1	1	1	2	2	0	II	1	1		80	60	44
159	75	1	1	6	2	2	2	1	2	0	I	2	2		100	80	98
160	55	1	1	8	2	2	2	1	2	0	IV	1	1	2	100	100	140
161	65	2	2	3	2	1	1	2	2	0	III	1	1	0.3	80	60	63
162	60	1	1	5	2	1	1	1	2	0	III	2	2	0.3	80	73.3	55
163	75	1	1	7	2	1	1	2	2	0	I	1	1	0.5	86.7	80	108
164	65	1	1	2	2	2	2	2	2	0	II	1	1	1.3	80	60	77
165	52	1	1	2	2	1	2	2	2	0	I	1	1	0.8	80	66.7	66
166	65	1	1	4	2	2	1	1	2	0	I	1	1	0.3	80	70	9
167	60	1	1	14	1	2	1	2	2	0	I	1	1	0.8	90	80	55
168	55	2	2	6	2	1	1	2	2	0	I	2	2	0.7	80	60	3

ANNEXURE VI

MASTER CHART

id	age	sex	cpc	duration	dur12hrs	dm	htn	smk	dyl	fh	klp	ecg	site	stemax	pretpte	posttpte	precqtd
169	42	1	1	24	1	1	1	2	2	0	I	2	2	0.3	80	80	18
170	65	1	1	5	2	1	1	2	2	0	I	1	1	0.4	85	80	39
171	50	1	1	6	2	2	2	2	2	0	I	2	2		100	60	26
172	53	1	1	24	1	2	1	2	2	0	I	1	1		110	80	76
173	35	1	1	8	2	2	2	1	2	0	I	1	1		84	80	26
174	70	1	1	23	1	1	2	2	2	0	I	1	1		80	90	106
175	54	1	1	4	2	2	2	2	2	0	I	2	2		100	80	65
176	34	1	1	3	2	2	2	1	2	0	I	1	1	0.2	80	57.1	115
177	73	1	1	12	1	1	2	2	2	0	I	2	2	0.2	96	75	80
178	49	1	1	1	2	2	2	1	2	0	I	1	1	1.6	86.7	80	26
179	59	2	1	2	2	1	1	2	2	0	I	1	1	1.4	80	60	46
180	41	1	1	18	1	1	1	2	2	0	I	2	2	0.6	93.3	73.3	62
181	80	1	1	5	2	2	2	1	2	0	I	2	2		100	80	95
182	47	1	1	10	2	2	2	2	2	0	I	1	1	2	140	100	24
183	61	1	1	3	2	2	1	2	2	0	I	2	2	0.3	77.5	76.7	76
184	77	1	1	2	2	1	1	1	2	0	I	2	2	0.2	80	80	139
185	47	1	1	4	2	2	2	1	2	1	II	1	1		100	80	110
186	62	1	1	4	2	1	2	2	2	0	IV	1	1	1.3	104	100	156
187	55	2	1	13	1	1	1	2	2	0	II	1	1	1.1	88	80	105
188	67	2	1	4	2	2	1	2	2	0	II	1	1	0.4	80	60	38
189	53	2	1	2	2	1	2	2	2	0	I	1	1	0.5	80	73.3	46
190	57	2	1	16	1	2	1	2	2	0	I	2	2		84	80	49
191	45	1	1	6	2	2	2	1	2	0	I	2	2	0.3	90	80	10
192	77	1	1	10	2	1	1	2	2	0	I	2	2	0.2	80	66.7	4
193	47	2	1	28	1	2	2	2	2	0	I	2	2	0.3	80	60	53
194	60	1	1	3	2	2	2	2	2	0	I	2	2	0.3	80	70	8
195	55	2	1	4	2	1	2	2	2	0	I	2	2	0.3	110	100	32
196	52	1	1	16	1	1	1	2	2	0	I	1	1		80	90	83
197	71	2	2	25	1	1	1	2	2	0	II	1	1	2.2	80	65	9
198	70	2	1	24	1	1	1	2	2	0	I	2	2	0.5	80	80	62
199	48	1	1	3	2	2	1	1	2	0	II	1	1	2.5	84	80	17
200	60	2	1	5	2	2	1	2	2	0	I	2	2	1.4	100	90	4
201	55	1	1	11	2	2	1	1	2	0	I	2	2		80	80	40
202	52	1	1	2	2	1	1	1	2	0	I	2	2	2	80	60	5
203	38	1	1	12	1	2	2	1	2	0	I	1	1		120	70	108
204	51	1	1	3	2	1	2	2	2	0	I	1	1	0.9	80	76	40
205	48	1	1	10	2	2	1	1	2	0	II	1	1	0.9	86.7	80	9
206	68	2	1	10	2	2	1	2	2	0	I	1	1	2	80	60	47
207	32	1	1	4	2	2	2	1	2	0	II	1	1	2	80	70	47
208	80	2	1	8	2	1	2	2	2	0	IV	2	2	0.5	104	100	150
209	35	1	1	6	2	2	2	1	2	0	IV	1	1	3	90	80	125
210	60	1	1	3	2	1	1	2	2	0	I	2	2	0.7	100	80	37

ANNEXURE VI

MASTER CHART

id	age	sex	cpc	duration	dur12hrs	dm	htn	smk	dyl	fh	klp	ecg	site	stemax	pretpte	posttpte	precqtd
211	60	1	1	5	2	2	1	1	2	0	II	1	1	2.2	80	73.3	29
212	65	1	1	3	2	2	1	2	2	0	II	2	2	0.9	93.3	76.7	112
213	50	1	1	2	2	2	2	2	2	0	I	2	2	1.9	100	86.7	19
214	54	1	1	5	2	1	2	2	2	0	I	2	2	0.3	80	76.7	41
215	53	1	1	5	2	2	2	1	2	0	I	2	2	0.8	100	66.7	44
216	38	1	1	1	2	2	2	1	2	0	I	1	1	2.2	100	66.7	22

ANNEXURE VI

MASTER CHART

id	postcqtd	lvef	inv	ves	Pretimi	Posttimi	Pretmp	Posttmp	stent	sized	sizel	n30dm	n30dhf	n30dary	TpTediff	cQtddiff
1	170	48	1	3	0	2	0		2	3	24	1	2	2	30	-56
2	32	42	2									1	2	2	27.5	42
3	49	37	1	1	0	2	0	1	2	3	18	1	2	2	33.3	17
4	70	53	3	1	0	2	1	1	2	3.5	23	1	2	2	40	-4
5	50	58	3	1	1	2	2	2	2	3.5	24	1	2	2	29	-7
6	62	36	1	1	0	1			2	3.5	21	1	2	2	4	-27
7	40	40	2									1	2	2	24	-13
8	144	39	2									1	2	2	5	-121
9	20	54	1	3	0	2			2	3.5	18	1	2	2	30	-8
10	32	42	3	2	0	2	0	1	2	3.5	14	1	2	2	30	1
11	74	46	1	2	0	2	1		0			1	2	2	0	39
12	51	54	1	1	0	2			2	3	18	1	2	2	20	-16
13	48	49	2									1	2	2	6.7	11
14	116	32	2	1								1	2	2	-13.3	-83
15	76	54	1	1	0	2			2	3.5	16	1	2	2	50	-30
16	125	32	3	2	0	2	1	1	1	3	18	1	2	2	0	-104
17	44	51	3	1	0	1	0	0	0			1	2	2	43.3	-28
18	86	53	2									1	2	2	40	-12
19	34	50	1	2	0	2	0	1	1	3	18	1	2	2	21.3	-6
20	50	35	1	2	0	2	0	0	2	2.5	18	2	1	2	-2.8	-46
21	26	37	1	1	0	2	0	1	1	3.5	18	1	2	2	16.7	31
22	60	33	3	1	0	2	1	1	1	2.75	18	1	2	2	8	4
23	132	46	3	3	0	2	0	1	2	2.75	12	1	2	2	10	-76
24	98	44	3									1	2	2	13.3	-57
25	223	25	2									2	1	2	-6.7	-25.2
26	54	58	1	1	0	2	0		0			1	2	2	-27	-10
27	45	42	1	3	0	2	0	0	2	2.5	33	1	2	2	0	-13
28	44	37	2	3								1	2	2	0	-18
29	9	48	1	1	0	2	1	1	2	3	18	1	2	2	11.4	2
30	55	32	1	2	0	2	0	1	1	2.75	18	1	2	2	6.7	9
31	23	53	1	2	0	2	0	1	2	3.5	28	1	2	2	13.4	-7
32	109	49	2									1	2	2	54	-29
33	65	44	1	2	0	2	0	1	1	3	26	1	2	2	17.5	1
34	14	33	1	2	0	2	1		1	3	21	1	2	2	11.9	-4
35	99	35	2									1	2	2	10	-1
36	46	49	1	3	0	2			2	2.5	18	1	2	2	20	-3
37	60	57	3	1	1	2	1	2	1	3.5	18	1	2	2	50	29
38	100	37	3	1	0	2	0	1	1	3	18	1	2	2	0.7	15
39	34	43	1	1	0	2	0	2	2	3.5	16	1	2	2	32	16
40	37	43	2									1	2	2	20	-9

ANNEXURE VI

MASTER CHART

id	postcqtd	lvef	inv	ves	Pretimi	Posttimi	Pretmp	Posttmp	stent	sized	sizel	n30dm	n30dhf	n30dary	TpTediff	cQtddiff
41	95	43	1	1	0	2	0	2	2	3	24	1	2	2	6.6	-31
42	48	54	2									1	2	2	-3.3	-19
43	101	64	1	2	0	2	2	2	1	3	32	1	2	2	20	-26
44	95	44	2									1	2	2	0	-35
45	87	44	2									1	2	2	6	13
46	87	51	2									1	2	2	-6.7	-62
47	90	32	1	1	0	2	0	1	2	2.75	21	1	2	2	19	-18
48	55	49	1	1	0	2	2	2	1	2.75	32	1	2	2	0	5
49	30	49	1	3	0	2	0	2	1	3.5	15	1	2	2	10	20
50	25	40	2									1	2	2	11	-11
51	38	38	2									1	2	2	30	34
52	100	39	1	3	1	2	0	1	0			1	2	2	12	20
53	50	45	2									1	2	2	0	-36
54	20	60	2	3								1	2	2	30	20
55	68	53	3	1	0	2	1		0			1	2	2	5.7	-34
56	108	44	2									1	2	2	20	25
57	150	49	2	3								1	2	2	50	-41
58	19	46	3	2	0	2	0		1	2.75	15	1	2	2	20	-1
59	70	50	2									1	2	2	24.2	-54
60	18	36	1	1	0	2	0	2	2	4	16	1	2	2	20	-8
61	98	42	2									1	2	2	0	-8
62	90	51	2									1	2	2	20	-40
63	92	48	1	1	0	2	2	2	2	2.75	12	1	2	2	1.1	-80
64	116	37	1	3	0	2	0	2	2	3.5	18	1	2	2	40	14
65	74	45	3	1	0	2	0	1	1	3.5	25	1	2	2	10.7	-12
66		30	2									2	2	2		
67	70	56	1	3	0	2	0		1	3.5	40	1	2	2	20	-30
68	70	37	2									1	2	2	30	40
69	60	38	1	1	0	2	0		2	3	21	1	2	2	10	50
70	29	58	3	3								1	2	2	20	14
71	3	41	1	1	0	2	1		2	3	23	1	2	2	44.8	8
72	118	43	3	1	0	2	0		1	2.75	20	1	2	2	44.2	-49
73	41	45	2	3								1	2	2	8	53
74	55	43	1	2	0	2	0	2	2	2.75	23	1	2	2	7.5	-54
75	148	62	2									1	2	2	0	-81
76	46	41	1	1	0	2	0	1	2	3	24	1	2	2	16	69
77	67	49	1	2	0	2	2	2	2	2.5	28	1	2	2	35	13
78	123	40	1	2	0	2	0		2	2.75	18	1	2	2	-10	-86
79	4	41	3	2	0	2	0	2	2	3	38	1	2	2	0	9
80	35	47	3	3	0	2			2	2.75	18	1	2	2	20	6
81	105	45	3	1	1	2	1	2	2	3.5	18	1	2	2	8.6	-15
82	26	44	1	1	0	2	0	1	2	3	18	1	2	2	6.7	49

ANNEXURE VI

MASTER CHART

id	postcqtd	lvef	inv	ves	Pretimi	Posttimi	Pretmp	Posttmp	stent	sized	sizel	n30dm	n30dhf	n30dary	TpTediff	cQtddiff
83	21	40	3	1	0	2	0	0	2	3.5	24	2	2	2	40	-11
84	25	32	1	1	0	2	0	2	1	3.5	13	1	2	2	13.3	21
85	15	37	3	1	0	2	0	2	1			1	2	2	15	30
86	12	56	1	2	0	2	0		2	2.5	18	1	2	2	-13.3	3
87	16	39	2									1	2	2	20	18
88	117	39	1	1	0	2	0	1	2	4	23	1	2	2	13.3	-89
89	31	22	1	3	0	2	0	0	2	2.75	23	2	1	2	0	-1
90	23	49	1	2	0	2	1	2	2	2.75	16	1	2	2	13.3	-4
91	58	49	1	1	0	2	0	2	1	3.5	22	1	2	2	15	-12
92	69	28	1	3	0	2			0			1	1	2	90	-58
93	53	46	3	3	0	2	1	2	1	2.75	12	1	2	2	14.2	-42
94	57	56	3	2	0	2	0	1	1	3.5	26	1	2	2	40	-15
95	122	40	1	1	0	2	0	1	2	3	21	1	2	2	16	-110
96	73	51	3	2	0	2			2	3	40	1	2	2	26.6	-63
97	74	41	3	1	0	2	0	2	1	3	18	1	2	2	5	-46
98	40	39	1	1	0	2	0	1	2	3.5	24	1	2	2	16.7	0
99	54	34	1	1	0	2			2	3	28	1	2	2	8	-21
100	80	38	1	2	0	2	0	2	2	2.75	40	1	2	2	16.7	-23
101	28	52	1	2	0	2	0		1	3	23	1	2	2	15.3	32
102	48	59	1	3	0	2	0		1	3	23	1	2	2	16.7	-21
103	90	45	1	3	0	2	0	1	1	3	18	1	2	2	6.7	-22
104	48	34	1	2	0	2	0		2	3	16	1	2	2	6.7	-32
105	55	47	3	2	1	2	0	0	1	3.5	18	1	2	2	8.3	-9
106	76	51	1	1	0	2	0	1	0			1	2	2	21.7	-4
107	80	46	3	1	0	2	1	1	2	3.5	18	1	2	2	24.2	-48
108	45	41	3	2	1	1	0	0	1	2.75	23	1	2	2	0	-23
109	85	48	3	1	1	2	1		1	3.5	28	1	2	2	6	-23
110	66	52	3		2	2	2		0			1	2	2	11.5	6
111	40	54	3	1	1	2	1		1	2.75	28	1	2	2	2.9	20
112	3	35	2									1	2	2	5	27
113	21	45	1	2	0	2	0		2	3	32	1	2	2	13.3	46
114	4	41	2									1	2	2	0	8
115	76	43	2	1								1	2	2	25.7	-10
116	44	39	2									1	2	2	10.7	11
117	82	38	1	1	0	2	1	2	1	3	12	1	2	2	20	-26
118	38	48	1	2	0	2			2	3.5	18	1	2	2	13.3	14
119	80	38	3	3	1	2	0	1	2	3.5	40	1	2	2	18.3	10
120	65	46	1	2	1	2	0	2	2	3	33	1	2	2	10	-45
121	75	54	2									1	2	2	12.5	-26
122	48	38	1	3	0	1	0		2	3	23	1	2	2	12	-27
123	12	43	1	3	0	2	0	1	2	3.25	18	1	2	2	13.3	3
124	11	56	1	1	0	2	0	2	2	3.5	18	1	2	2	25	4

ANNEXURE VI

MASTER CHART

id	postcqtd	lvef	inv	ves	Pretimi	Posttimi	Pretmp	Posttmp	stent	sized	sizel	n30dm	n30dhf	n30dary	TpTediff	cQtddiff
125	117	40	1	1	0	2	2	2	2	3	18	1	2	2	26	-21
126	60	42	1	1	0	2	0		2	3	23	1	2	2	16	-10
127	84	39	3	2	0	2	0	2	2	3	12	1	2	2	13.4	-30
128	122	33	1	2	0	2	2	2	1	3	23	1	2	2	20	-43
129	54	44	1	1	0	0	0		0			1	2	2	10.9	4
130	38	57	2									1	2	2	5	-15
131	106	35	1	3	0	2	0	1	0			1	2	2	6.7	12
132	17	58	2									1	2	2	52	69
133	48	40	1	1	0	1	2	2	1	3.5	23	1	2	2	13	-9
134	44	56	2									1	2	2	5	39
135	35	33	2									1	2	2	6.7	31
136	24	50	3	1	0	2	0	1	1	3.5	23	1	2	2	40	26
137	76	56	3	1	0	2	1	1	1	2.75	12	1	2	2	20	8
138	109	48	1	3	0	2	0	1	2	3	15	1	2	2	26.7	-5
139	112	34	1	2	0	2	0		1	3	23	1	2	2	3.3	-32
140	65	54	1	3	0	2	0	2	2	3	40	1	2	2	6	24
141	39	42	2	1								1	2	2	13.3	20
142	32	52	1	2	0	2	0	2	2	3.5	18	1	2	2	13.3	-12
143	70	41	1	1	0	2	0		2	3	24	1	2	2	5	32
144	129	40	2	2					1			2	2	1	5	-83
145	44	40	1	3	0	2	0		1	2.75	15	1	2	2	8.3	-18
146	76	43	2									1	2	2	2.28	11
147	117	42	3	1	0	2	0		1	2.5	18	1	2	2	6.7	-21
148	32	55	3	2	0	2	0		2	2.75	18	1	2	2	6.7	18
149	27	40	1	1	0	2	0	1	2	3	20	1	2	2	8	-14
150	40	37	3	2	0	2	1	1	2	3	24	1	1	2	0	42
151	111	54	1	1	1	2			2	2.75	28	1	2	2	5	-109
152	68	45	3	1	0	2	0	0	1	3	15	1	2	2	13.3	-63
153	37	40	1	1	0	2	0	1	2	3	16	1	2	2	0	1
154	69	50	2									1	2	2	-6.7	-65
155	213	30	2									1	2	2	40	-57
156	72	34	1	3	0	2			1	3	20	1	2	2	30	-46
157	34	38	1	1	0	2			1	3	15	1	2	2	10	8
158	64	48	1	1	0	2			2	3.5	42	1	2	2	20	-20
159	85	42	1	3	0	2			1	3.5	28	1	2	2	20	13
160	22	42	1	3	0	1	0	0			2	1	2	0	118	
161	92	41	2									1	2	2	20	-29
162	54	40	2	3								1	2	2	6.7	1
163	138	51	2									1	2	2	6.7	-30
164	52	43	2									1	2	2	20	25
165	38	46	1	2	0	2	0		2	2.75	38	1	2	2	13.3	28
166	115	46	2									1	2	2	10	-106

ANNEXURE VI

MASTER CHART

id	postcqtd	lvef	inv	ves	Pretimi	Posttimi	Pretmp	Posttmp	stent	sized	sizel	n30dm	n30dhf	n30dary	TpTediff	cQtddiff
167	134	42	1	1	0	2	0	1	2	2.75	24	1	2	2	10	-79
168	28	56	1	2	0	2	0	2		2	2	2	20	-25		
169	27	43	1	3	0	2	2	2	2	3	32	1	2	2	0	-9
170	37	30	1	3	0	2	0	0	1	3	30	2	2	2	5	2
171	107	55	1	2	0	2			2	3	16	1	2	2	40	-81
172	63	46	3	2	1	2			1	3	28	1	2	2	30	13
173	22	41	3	1					0			1	2	2	4	4
174	70	39	1	2	0	2			2	2.75	28	1	2	2	-10	36
175	38	51	2									1	2	2	20	27
176	56	45	3	3	0	2	0		2	3	24	1	2	2	22.9	59
177	177	49	2									1	2	2	21	-97
178	94	61	1	1	0	2	0	1	2	3	23	1	2	2	6.7	-68
179	192	41	1	3	0	2	0		2	2.75	24	1	2	2	20	-146
180	172	56	1	1	0	2			2	3	12	1	2	2	20	-110
181	68	42	1	3	0	2			2	3	32	1	2	2	20	27
182	29	56	1	2	0	2	0	1	2	3	20	1	2	2	40	-5
183	180	44	1	2	0	2	1	2	1	2.75	24	1	2	2	0.8	-104
184	239	45	1	3	0	2	0		1	3.5	22	1	2	2	0	-100
185	76	37	1	1	0	2			2	3	32	1	2	2	20	34
186	46		2									2	1	1	4	110
187	25	54	1	1	0	2	0	0	2	3	21	1	2	2	8	80
188	33	40	2	3								1	2	2	20	5
189	55	35	2									1	2	2	6.7	-9
190	20	54	1	1	0	2	0	1	2	3.5	18	1	2	2	4	29
191	75	56	3	1	1	2	0	0	2	3.5	28	1	2	2	10	-65
192	40	56	1	2	0	2	2	2	1	2.75	23	1	2	2	13.3	-36
193	41	50	1	1	0	2	0	1	1	2.75	23	1	2	2	20	12
194	48	46	1	1	0	2			1	2.5	14	1	2	2	10	-40
195	27	54	3	2	1	2	1	2	1	2.75	18	1	2	2	10	5
196	76	54	1	1	0	2			2	2	18	1	2	2	-10	7
197	37	42	1	1	0	2	0	0	0			1	2	2	15	-28
198	29	50	1	1	0	2	0	1	2	3	33	1	2	2	0	33
199	41	41	1	2	0	2	0	0	2	3	24	1	2	2	4	-24
200	33	45	1	2	0	2	0		1	3.5	26	1	2	2	10	-29
201	130	36	2									1	2	2	0	-90
202	34	54	1	2	0	2	0	0	2	3.5	18	1	2	2	20	-29
203	86	34	3	1	0	2			1	3.5	26	1	2	2	50	22
204	41	42	2									1	2	2	4	-1
205	71	38	1	1	0	2	0		2	3	21	1	2	2	6.7	-62
206	60	49	1	2	0	2	0	1	2	3.5	24	1	2	2	20	-13
207	50	45	1	1	0	2	2	2	2	3.5	18	1	2	2	10	-3
208	89	29	1	3	0	1	0			3	23	1	2	1	4	61

ANNEXURE VI

MASTER CHART

id	postcqtd	lvef	inv	ves	Pretimi	Posttimi	Pretmp	Posttmp	stent	sized	sizel	n30dm	n30dhf	n30dary	TpTediff	cQtddiff
209	85	20	1	2	0	0	0		2			2	1	2	10	40
210	129	54	2									1	2	2	20	-92
211	61	50	1	1	0	2	2	2	2	2.75	12	1	2	2	6.7	-32
212	5	45	2									1	2	2	16.6	107
213	88	55	1	2	0	2	0	1	2	3	18	1	2	2	13.3	-69
214	49	56	1	1	0	2	0	2	2	3	23	1	2	2	3.3	-8
215	55	55	3	1	0	1			1	2.75	26	1	2	2	33.3	-11
216	55	26	3	1	1	2			2	3	28	1	2	2	33.3	-33